MAY 3 1926

Medical Lib


# THE BOSTON Medical and Surgical JOURNAL

**Owned and Published by the MASSACHUSETTS MEDICAL SOCIETY**

OFFICIAL ORGAN OF THE NEW ENGLAND SURGICAL SOCIETY, THE BOSTON SURGICAL SOCIETY, INC., THE NEW ENGLAND PEDIATRIC SOCIETY, AND THE NEW HAMPSHIRE SURGICAL CLUB

Volume 194, Number 17 | APRIL 29, 1926 | Published Weekly in Boston
$6 per Annum, 25c per Copy | | at 126 Massachusetts Avenue


## CONTENTS


**ORIGINAL ARTICLES**

Reticulated Red Cells—Their Clinical Significance. *William Dameshek* ..... 759

A Review of Our Present Knowledge of the Pathology and Roentgen Diagnosis of Periapical Tooth Infection. *Kurt H. Thoma* ..... 768

**NEW ENGLAND PEDIATRIC SOCIETY**

Chronic Fatigue in the School Child. A Psychophysiologic Study. *Max Seham* ..... 770

**MEDICAL PROGRESS**

Progress in the Study and Treatment of Cardiovascular Disease in 1925. *T. Duckett Jones* and *Howard B. Sprague* ..... 777

**CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL**

Case 12171, Sympathectomy for Angina Pectoris in a Woman of Twenty-Two. *Paul D. White* and *Edward P. Richardson;* Case 12172, *Arthur B. Lyons;* Case 12173, *Richard H. Miller* ..... 790


*(Contents continued on next page)*

THE NEXT ANNUAL MEETING OF THE MASSACHUSETTS MEDICAL SOCIETY WILL BE HELD IN SPRINGFIELD ON TUESDAY AND WEDNESDAY, JUNE 8 AND 9, 1926.

For list of Officers of the Society and of the District Medical Societies, see advertising page ii.


# *Fourth Edition* --- DeLee's Obstetrics

## WITH $15,000 WORTH OF ILLUSTRATIONS

IT is said that Dr. DeLee's book rings true. Those who know how it was written are not surprised at this. It rings true because it *is* true; because it is not a compilation, because it was literally written at the bedside.

You will find the work extremely practical throughout, Dr. Lee's aim having been to produce a book that would meet fully every need of the practitioner as well as the obstetrician. For this reason *diagnosis* and the relations of obstetric conditions and accidents to general medicine, surgery, and the specialties are featured.

Regarding *treatment:* You get here the very latest advances in this field, and you can rest assured every method of treatment, every step in operative technic is just right.

The *descriptive legends* under the illustrations are unusually full, and, by studying the pictures serially with their detailed legends, you are better able to follow the operations than by referring to the pictures from a distant text.

"I consider Dr. DeLee's work one of the best books on obstetrics published in the English language, and will take great pleasure in recommending it."—*Dr. John A. McGlinn, University of Pennsylvania.*

*Principles and Practice of Obstetrics.* By JOSEPH B. DELEE, M.D., Professor of Obstetrics at the Northwestern University Medical School, Chicago. Large octavo of 1123 pages, with 1128 illustrations, 201 in colors. Cloth, $12.00 net.

**W. B. SAUNDERS COMPANY** — **Philadelphia and London**


Entered at the Postoffice at Boston as Second Class Matter

# CONTENTS

*(Continued)*

EDITORIALS
Massachusetts Medical Benevolent Society ..... 803
Delayed Justice ..... 803
The Water Supply for Metropolitan Boston ..... 804
This Week's Issue ..... 804

BOOK REVIEW
Light and Health. M. Luckiesh ..... 808

LEGISLATIVE NOTE ..... 805

MISCELLANY
Connecticut Department of Health—Morbidity Report for the Week Ending April 17, 1926 ..... 807
Fruit Made Poisonous by Sprays ..... 805
Massachusetts Department of Public Health—Diseases Reported for the Week Ending April 17, 1926 ..... 805
Resume of Communicable Diseases Reported in Massachusetts, March, 1926 ..... 806
Universal Antitoxin ..... 805

NEWS ITEMS ..... 807

RECENT DEATH ..... 805

REPORTS AND NOTICES OF MEETINGS ..... 807

**National Antacid Powder**
Dr. Sippy's Formula
For GASTRIC ULCER
Composed of
Bismuth Subcarbonate ..... 1 part
Sodium Bicarbonate ..... 2 parts
Calcium Carbonate (precip.) ..... 2 parts
Magnesium Oxide Light ..... 2 parts
Unflavored
Samples and Literature
Boston Office
THE NATIONAL DRUG CO.
53 State Street, Boston, Mass.
At all druggists in 2 oz. packages

**SPENCER**
**CORSET SHOP**
Individual fitting our specialty.
**Mrs. Mary L. H. Wood**
Successor to Miss B. W. Logan
462 Boylston St., Boston, Mass.
Room 210
Telephone: Back Bay 5824

# The Massachusetts Medical Society

## OFFICERS ELECTED BY THE COUNCIL, JUNE 9, 1925, FOR 1925-1926

PRESIDENT: JAMES S. STONE, 286 Marlborough St., Boston.
VICE-PRESIDENT: MICHAEL F. FALLON, 390 Main St., Worcester.
SECRETARY: WALTER L. BURRAGE, 182 Walnut St., Brookline.
TREASURER: ARTHUR K. STONE, Auburn St., Framingham Center.
LIBRARIAN EMERITUS: EDWIN H. BRIGHAM, 45 Forest St., Wellesley Hills.

### STANDING COMMITTEES FOR 1925-1926

ON ARRANGEMENTS
John Rock, L. S. McKittrick, W. T. S. Thorndike, James Hitchcock, E. P. Hayden, H. Q. Gallupe.

ON PUBLICATIONS AND SCIENTIFIC PAPERS
E. W. Taylor, R. B. Osgood, F. T. Lord, R. M. Green, A. C. Getchell.

ON MEMBERSHIP AND FINANCE
D. N. Blakely, Algernon Coolidge, Samuel Crowell, Gilman Osgood, Homer Gage.

ON ETHICS AND DISCIPLINE
Henry Jackson, David Cheever, W. D. Ruston, S. F. McKeen, W. C. Keith.

ON MEDICAL EDUCATION AND MEDICAL DIPLOMAS
C. F. Painter, J. F. Burnham, A. G. Howard, R. L. De Normandie, H. P. Stevens.

ON STATE AND NATIONAL LEGISLATION
J. S. Stone, E. H. Stevens, F. E. Jones, T. J. O'Brien, J. H. Birnie.

ON PUBLIC HEALTH
Victor Safford, Annie L. Hamilton, E. F. Cody, R. I. Lee, T. F. Kenney.

ON PUBLIC INSTRUCTION
A. P. Merrill, Kendall Emerson, W. P. Bowers, F. W. Snow, G. C. Shattuck, W. H. Robey, R. I. Lee.

## OFFICERS OF THE DISTRICT MEDICAL SOCIETIES

ELECTED BY THE DISTRICT MEDICAL SOCIETIES BETWEEN APRIL 15 AND MAY 15, 1925

BARNSTABLE—*President*, E. F. Curry, Sagamore; *Vice-President*, F. O. Cass, Provincetown; *Secretary*, P. P. Henson, South Yarmouth; *Treasurer*, H. B. Hart, Yarmouthport; *Librarian*, E. E. Hawes, Hyannis.

BERKSHIRE—*President*, Nathan Finkelstein, Pittsfield; *Vice-President*, George F. MacKay, Dalton; *Secretary*, A. P. Merrill, Pittsfield; *Treasurer*, C. T. Leslie, Pittsfield.

BRISTOL NORTH—*President*, A. S. MacKnight, Attleboro; *Vice-President*, F. H. Dunbar, Mansfield; *Secretary*, J. L. Murphy, Taunton; *Treasurer*, R. D. Dean, Taunton.

BRISTOL SOUTH—*President*, E. F. Curry, Fall River; *Vice-President*, S. E. Donovan, New Bedford; *Secretary*, G. E. Borden, Fall River; *Treasurer*, G. E. Borden, Fall River.

ESSEX NORTH—*President*, R. C. Hurd, Newburyport; *Vice-President*, R. V. Baketel, Methuen; *Secretary*, J. Forrest Burnham, Lawrence; *Treasurer*, E. H. Ganley, Methuen.

ESSEX SOUTH—*President*, J. F. Donaldson, Salem; *Vice-President*, T. L. Jenkins, Topsfield; *Secretary*, R. E. Stone, Beverly; *Treasurer*, Andrew Nichols, 3rd, Danvers; *Librarian*, C. M. Cobb, Lynn.

FRANKLIN—*President*, F. A. Millett, Greenfield; *Vice-President*, P. F. Leary, Turners Falls; *Secretary*, Charles Moline, Sunderland; *Treasurer*, Charles Moline, Sunderland.

HAMPDEN—*President*, W. C. Leary, Springfield; *Vice-President*, G. H. Janes, Westfield; *Secretary*, H. L. Smith, Springfield; *Treasurer*, H. L. Smith, Springfield.

HAMPSHIRE—*President*, F. E. Dow, Northampton; *Vice-President*, W. B. Segur, Enfield; *Secretary*, L. O. Whitman, Northampton; *Treasurer*, L. O. Whitman, Northampton; *Librarian*, F. H. Smith, Hadley.

MIDDLESEX EAST—*President*, C. R. Henderson, Reading; *Vice-President*, R. R. Stratton, Melrose; *Secretary*, A. R. Cunningham, Winchester; *Treasurer*, Richard Dutton, Wakefield; *Librarian*, G. W. Nickerson, Stoneham.

MIDDLESEX NORTH—*President*, J. F. Boyle, Lowell; *Vice-President*, M. L. Alling, Lowell; *Secretary*, T. A. Stamas, Lowell; *Treasurer*, E. J. Clark, Lowell; *Librarian*, P. J. Meehan, Lowell.

MIDDLESEX SOUTH—*President*, G. L. West, Newton Center; *Vice-President*, A. W. Dudley, Cambridge; *Secretary*, S. M. Biddle, Cambridge; *Treasurer*, Edward Mellus, Newton.

NORFOLK—*President*, J. E. Fish, Canton; *Vice-President*, C. W. Macdonald, Roxbury; *Secretary*, F. S. Cruickshank, Brookline; *Treasurer*, G. W. Kaan, Brookline.

NORFOLK SOUTH—*President*, W. G. Curtis, Wollaston; *Vice-President*, D. A. Bruce, Atlantic; *Secretary*, N. R. Pillsbury, South Braintree; *Treasurer*, N. R. Pillsbury, South Braintree; *Librarian*, N. R. Pillsbury, South Braintree.

PLYMOUTH—*President*, J. H. Drohan, Brockton; *Vice-President*, A. C. Smith, Brockton; *Secretary*, L. B. Packard, Brockton; *Treasurer*, L. B. Packard, Brockton; *Librarian*, J. H. Weller, Bridgewater.

SUFFOLK—*President*, C. M. Smith, Boston; *Vice-President*, G. R. Minot, Boston; *Secretary*, Arthur H. Crosbie, Boston; *Treasurer*, J. C. Rock, Boston; *Librarian*, E. C. Streeter, Boston.

WORCESTER—*President*, R. P. Watkins, Worcester; *Vice-President*, E. H. Trowbridge, Worcester; *Secretary*, P. H. Cook, Worcester; *Treasurer*, G. O. Ward, Worcester; *Librarian*, A. C. Getchell, Worcester.

WORCESTER NORTH—*President*, J. A. Barton, Fitchburg; *Vice-President*, J. P. Norton, Fitchburg; *Secretary*, C. H. Jennings, Fitchburg; *Treasurer*, F. H. Thompson, Jr., Fitchburg.

THE BOSTON
Medical and Surgical
JOURNAL

VOLUME 194 APRIL 29, 1926 NUMBER 17

## ORIGINAL ARTICLES

### THE RETICULATED RED CELLS—THEIR CLINICAL SIGNIFICANCE*

BY WILLIAM DAMESHEK, M.D.

SINCE the introduction of vital staining methods of the blood by Ehrlich[1] at the end of the last century, the significance of the reticulated cells, though more or less realized, has not yet, in my opinion, been fully sensed. The reticulate count, once made quite frequently, has now been (except in a few laboratories) discarded and forgotten. Here at the Boston City Hospital, in the Blood Laboratory and Clinic which was organized in 1919, the reticulate count has been done as a routine measure on all patients. Considerable data have thus been accumulated and can now be viewed in their proper perspective.

So-called vital staining consists in mixing a drop of freshly drawn blood with a bit of stain, and then observing the blood before it has dried. Of course, the staining is not truly "vital", since either the living cell does not take up the stain, or if it does, the cell is thereby damaged to a greater or less extent. Accordingly, the process has been called a "post-vital" or "supravital" staining. Needless to say, some of the characteristics (if not all) that one sees with vital staining have been called necrobiotic phenomena, and have thereby been more or less discredited and disparaged. However, there is no doubt but that the staining is more "vital" and true than that of dried or otherwise fixed preparations, and of a great deal of value.

When methylene blue was first used in this way by Ehrlich, several of the red cells were seen to contain in their cytoplasm a bluish network. Later by the use of other stains such as neutral red (Pappenheim[2]), granules were occasionally seen especially in anemias, and finally, by the use of brilliant cresyl violet which was developed by the Italian Cesaris-Demel[2a], various structures such as granules, filaments, and networks were discerned. The name most commonly applied to these red cells which, when stained vitally, take on in parts a deep blue or violet coloring is, in Europe, the granulo-reticulo-filamentous cells. This name has in America been abbreviated to simply, "reticulated" cells.

Most authors have agreed that usually, the percentage of these cells is about one, slightly under or over; that in pernicious anemia, the percentage is often increased, and in congenital hemolytic anemia extremely high—often 30 to 40.

Why certain of the red cells of the blood should take on this coloration when stained vitally, and the significance of their number, have long been debatable questions. Reticulation has been called a necrobiotic phenomenon by some (Naegeli[3]), and by others a staining of intracellular lipoids (Pappenheim[4]). By others (Key[5]) the reticulum has been likened to the more or less debatable mitachrondria. Noting the large number of reticulated cells in hemolytic anemia, and also the markedly increased fragility of the red cells, it was but natural that earlier observers (Widal[6] *et al;* Chauffard and Fiessinger[7]) should have concluded that the reticulated cells were the fragile ones; this has since been disproved (Pepper and Peet[8]; Buckman and MacNaugher[9]).

Frankly, it is unknown why these cells are so stained. Physiologically, all are agreed that the reticulated cells represent the young growing red cells. Why these cells are not seen in the ordinary fixed preparation stained by ordinary means, is a mystery. Apparently, these youngsters are differentiated only by the delicate method of vital staining.

Again, what the relationship is between reticulated cells and the basically stippled cells of the fixed preparation, is a hotly debated subject. By numerous authors, the reticulated cells are the stippled cells of the fixed preparations; by numerous others, they are entirely different. This subject is, as can be seen, a very mooted and a very complicated one. Suffice it to say that both reticulation and stippling have been by most agreed upon as evidences of regeneration—reticulation showing normal regeneration, basic stippling, pathological or abnormal regeneration, due to toxic or other processes. It must be remembered that the reticulate count is made only in a vitally stained specimen, and that stippling is seen only in the fixed preparation. (For an excellent discussion of this subjects see Ferrata, A.[9a].)

*From the Blood Laboratory, Boston City Hospital.

TABLE I

PERNICIOUS ANEMIA—RETICULATE COUNT

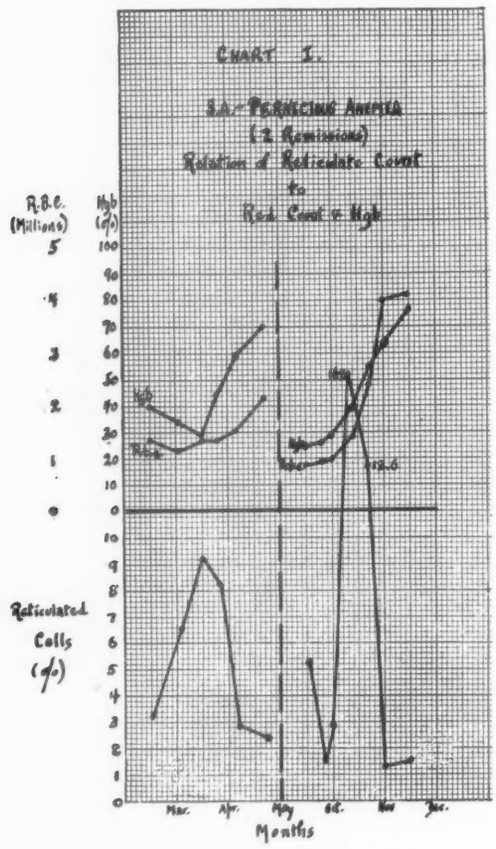
| Case | Initials | Sex | Age | Stage of Disease | Appearance time of Reticulosis before Remission | Reticulate Count | Hgb | Red cells (Millions) | Result |
|---|---|---|---|---|---|---|---|---|---|
| 1 | M. A. | ♀ | 60 | Relapse | | 2.6-1.2 | 33-28 | 1.1-1.6 | Death |
| 2 | R. A. | ♂ | 27 | Remission 1. | (Transfusion) | 29.6 | 14-82 | 0.5-3.9 | |
| | | | | Relapse | | 2.6-1.8 | 82-33 | 3.9-1.4 | |
| | | | | Remission 2. | (Transfusion) | 4.4-5.4 | 33-63 | 1.4-2.5 | |
| 3 | S. A. | ♂ | 58 | Remission 1. | Two weeks | 6.6-9.2-8.2 | 30-71 | 1.4-2.2 | |
| | | | | Relapse | | 1.4-2.8 | 25 | 0.9 | |
| | | | | Remission | One week | 16.2-12.6 | 25-78 | 0.9-4.1 | |
| 4 | L. A. | ♀ | 65 | Relapse | | 1.6-5.6 | 38-47 | 1.7-1.3 | Death in 4 mos. |
| 5 | E. B. | ♀ | 72 | Relapse | | 3.0-0.9 | 12-30 | 0.6-1.2 | Death |
| 6 | J. B. | ♂ | 61 | Remission 1. | Two weeks | 8.0 | 34-99 | 1.0-4.4 | |
| | | | | Relapse | | 0.6-2.0 | 99-41 | 4.4-1.5 | |
| | | | | Remission 2. | One week | 4.6 | 41-90 | 1.5-4.0 | |
| 7 | E. B. | ♂ | 60 | Remission | Two weeks | 8.2 | 68-85 | 1.5-2.8 | |
| 8 | A. B. | ♀ | 55 | Relapse | | 2.5-0.3 | 41-20 | 1.3-0.7 | Death |
| 9 | A. C. | ♀ | 30 | Remission | One week | 6.0-5.8 | 27-96 | 1.3-3.3 | |
| 10 | S. C. | ♂ | 58 | Remission | One week | 14.6 | 38-80 | 1.0-4.3 | |
| 11 | C. C. | ♀ | 72 | Relapse | | 2.7-0.2 | 25-29 | 0.8-1.3 | Death |
| 12 | K. D. | ♀ | 34 | Remission 1. | | 1.9-3.6 | 27-74 | 1.2-2.9 | |
| | | | | Relapse | | 3.8-1.0 | 74-28 | 2.9-0.9 | |
| | | | | Remission 2 | (Splenectomy) Two weeks | 10.2-8.8 | 45-86 | 1.8-3.5 | |
| 13 | N. D. | ♀ | 55 | Relapse | | 5.0-1.0 | 52-34 | 1.7-0.9 | Death |
| 14 | N. F. | ♂ | 48 | Relapse | | 0.5-1.8 | 68-34 | 2.1-1.1 | Death |
| 15 | J. J. F. | ♂ | 60 | Relapse | | 0.6-1.4 | 38-24 | 1.2-1.1 | Death |
| 16 | C. F. | ♀ | 60 | Relapse | | 3.8-1.0 | 47-15 | 1.3-0.5 | Death |
| 17 | W. F. | ♂ | 48 | Remission 1. | One week | 9.6 | **34-100** | **1.0-4.3** | |
| | | | | Relapse 1. | | 1.6-8 | 100-20 | 4.3-0.5 | |
| | | | | Remission 2. | (Splenectomy) | 8.8 | 20-64 | 0.5-2.4 | |
| | | | | Relapse 2. | | 1.2-2.8 | 64-41 | **2.4-1.4** | |
| | | | | Remission 3. | | 8.2 | 41-64 | 1.4-2.6 | |
| 18 | W. H. | ♂ | 52 | Remission | (Transfusion) | 15.4 | 41-70 | 2.0-2.2 | |
| | | | | Relapse | | 1.2-2.0 | 70-27 | 2.9-1.2 | Death |
| 19 | W. H. | ♂ | 54 | Relapse | | 3.4-0.3 | 50-15 | 1.6-0.9 | Death |
| 20 | A. H. | ♀ | 62 | Remission 1. | | 1.3-6.8 | 49-93 | 1.6-4.1 | |
| | | | | Relapse 1. | | 0.6-1.0 | 98-45 | 4.1-1.9 | |
| | | | | Remission 2. | (Transfusion) | 14-20 | 23-74 | **0.9-3.5** | |
| 21 | E. F. | ♀ | 65 | Remission | ? | Not done | 35-71 | 1.2-2.5 | |
| | | | | Relapse | | 5.4-0.8 | 71-35 | 2.5-1.2 | Death |
| 22 | T. G. | ♂ | 48 | Remission | (Transfusions) | 1.4 | 45-65 | 1.4-3.4 | |
| 23 | S. H. | ♂ | 62 | Remission | One week | 7.4-8.0 | 29-86 | 1.3-2.4 | |
| | | | | Relapse | | 0.3-1.2 | 86-27 | 2.4-1.1 | Death |
| 24 | R. H. | ♂ | 59 | Remission | One week | 7.2-13.0 | 30-71 | 1.2-2.3 | |
| 25 | S. J. | ♀ | 52 | ? Remission | Two weeks | 9.4 | 42-62 | 1.4-1.7 | |
| | | | | Relapse | | 0.6-2.2 | 62-24 | 1.7-0.9 | Death |
| 26 | I. L. | ♀ | 58 | Remission 1. | (Transfusion) | 2.8-3.8 | 34-61 | 1.3-3.0 | |
| | | | | Relapse 1. | | 3.8-1.2 | 61-25 | 2.4-0.8 | |
| | | | | Remission 2. | | 5.0 | 25-43 | 0.8-1.6 | |
| | | | | Relapse | | 1.6-8.0-0.9 | 50-38 | 1.8-1.6 | **Death** |
| 27 | A. L. | ♀ | 72 | Relapse | | 1.6-3.2 | 41-52 | 1.3-1.8 | Death |

TABLE I (*continued*)

| Case | Initials | Sex | Age | Stage of Disease | Appearance time of Reticulosis before Remission | Reticulate Count | Hgb | Red cells (Millions) | Result |
|---|---|---|---|---|---|---|---|---|---|
| 28 | F. M. | ♂ | 64 | Relapse | ..... | 0.6-0.1 | 13-19 | 0.7-1.0 | Death |
| 29 | C. M. | ♀ | 56 | Relapse 1. | ..... | 7.5-3.0 | 45-35 | 1.2-0.7 | |
| | | | | ? Remission | (Transfusion) | 2.0 | 35-60 | 0.7-2.2 | |
| | | | | Relapse 2. | ..... | 1.-2. | 60-29 | 2.2-0.8 | Death |
| 30 | M. L. | ♀ | 58 | Remission | One week | 11.0-14.2 | 36-67 | 1.1-2.3 | |
| 31 | R. McH. | ♀ | 42 | Remission | Ten days | 5.8-17.0 | 15-94 | 0.8-4.6 | |
| | | | | Relapse | ..... | 1.0-0.8 | 94-17 | 4.6-0.8 | Death |
| 32 | S. McI. | ♀ | 65 | Relapse 1. | ..... | 0.5-1.0 | 30-21 | 0.8-0.8 | |
| | | | | ? Remission | Two weeks | 7.8 | 19-38 | 0.9-1.1 | |
| | | | | Relapse 2. | ..... | 1.0 | 38-13 | 1.1-0.4 | |
| | | | | Remission 2. | ..... | 12.2 | 13-71 | 0.4-4.7 | |
| | | | | Relapse 3. | ..... | 3.7-2.6 | 71-56 | 4.7-2.5 | Death |
| 33 | J. McD. | ♂ | 52 | Remission 1. | Three days | 15.8 | 40-67 | 1.7-3.1 | |
| | | | | Relapse 1. | ..... | 0.9-1.6 | 67-32 | 3.1-0.9 | |
| | | | | Remission 2. | ..... | 4.8-7.8 | 32-71 | 1.2-3.5 | |
| | | | | Relapse | ..... | 1.2 | 71-50 | 3.5-2.6 | |
| 34 | A. McD. | ♀ | 58 | Relapse | ..... | 5.6-0.5 | 58-18 | 2.7-0.7 | Death |
| 35 | D. McS. | ♂ | 52 | Remission 1. | One week | 7.2 | 35-73 | 1.3-4.0 | |
| | | | | Relapse 1. | ..... | 1.8-1.6 | 74-34 | 2.9-1.0 | |
| | | | | Remission 2. | (Transfusion) | 25. | 47-77 | 1.8-2.6 | |
| | | | | Relapse 2. | ..... | 1.0 | 77-30 | 2.6-1.3 | |
| | | | | Remission 3. | (Splenectomy) | 1.0-2.6 | 39-96 | 1.3-5.0 | |
| 36 | M. McD. | ♀ | 56 | Remission | (Transfusion and Splenectomy) | 1-4 | 27-71 | 1.2-2.5 | |
| | | | | Relapse | ..... | 0.5-1.0 | 71-20 | 2.5-1.0 | |
| 37 | S. McC. | ♀ | 52 | Relapse 1. | ..... | 1.6-5.2 | 53-26 | 2.0-0.8 | |
| | | | | Remission 1. | One week | 8.0 | 26-100 | 0.8-5.4 | |
| | | | | Relapse 2. | ..... | 1-4 | 100-50 | 5.4-2.0 | |
| | | | | Remission 2. | One week | 5.2-6.0 | 50-85 | 1.9-3.3 | |
| 38 | E. McR. | ♀ | 68 | Remission | Two weeks | 6.6-20.1-10.2 | 23-91 | 1.0-5.2 | |
| 39 | E. M. | ♀ | 58 | ? Remission | ..... | 1.2-5.6 | 39-48 | 2.1-2.8 | |
| | | | | Relapse | ..... | 1.0-6.0 | 36-37 | 1.3-1.2 | Death |
| 40 | N. P. | ♀ | 48 | Remission 1. | (Transfusion) | 2-4 | 15-64 | 0.9-2.4 | |
| | | | | Relapse 1. | ..... | 1.0 | 64-46 | 2.4-1.7 | |
| | | | | Remission 2. | Four days | 12.6 | 46-69 | 1.7-2.2 | |
| | | | | Relapse 2. | ..... | 0.5 | 69-32 | 2.2-1.2 | Death |
| 41 | J. R. | ♂ | 38 | Remission | Two weeks | 6.4 | 22-70 | 0.9-4.0 | |
| | | | | Relapse | ..... | 0.5-0.2 | 70-15 | 2.2-0.6 | Death |
| 42 | E. S. | ♀ | 58 | Remission 1. | ? | 5.4 | 45-100 | 1.2-2.0 | |
| | | | | Relapse 1. | ..... | 1.4 | 100-35 | 2.0-1.0 | |
| | | | | Remission 2. | One week | 10.2 | 35-74 | 2.0-2.3 | |
| | | | | Relapse 2. | ..... | 0.5-0.2 | 70-15 | 0.7-0.4 | Death |
| 43 | W. S. | ♂ | 64 | Remission | ? | ? | 36-65 | 1.5-1.4 (?) | |
| 44 | S. T. | ♀ | 60 | Relapse | ..... | 1-4 | 16-40 | 0.4-1.6 | Death |
| 45 | J. E. S. | ♂ | 44 | Relapse | ..... | 5.4-0.8 | 35-18 | 0.9-0.7 | Death |
| 46 | M. W. | ♀ | 58 | Relapse | ..... | 2.8-0.6 | 58-17 | 2.7-0.8 | Death |
| 47 | A. W. | ♀ | 48 | Remission 1. | ..... | 3.2 | 55-84 | 1.9-3.2 | |
| | | | | Relapse 1. | ..... | 0.8-1.5 | 84-37 | 3.2-1.5 | |
| | | | | Remission 2. | One week | 10.5-13.7 | 37-59 | 1.5-2.6 | |
| 48 | S. W. | ♀ | 44 | Remission | Ten days | 9.6-13.4 | 47-92 | 1.4-3.0 | |
| 49 | E. W. | ♀ | 28 | Relapse 1. | ..... | 1.2 | 50-35 | 1.5-0.7 | |
| | | | | Remission 1. | One week | 8.0-26.2 | 35-99 | 0.7-4.5 | |
| | | | | Relapse 2. | ..... | 5.8-3.4 | 99-19 | 4.5-0.5 | |
| | | | | Remission 2. | (Transfusion) | 15.0 | 19-90 | 0.5-3.5 | |
| | | | | Relapse 3. | ..... | 0.7-4 | 90-14 | 3.5-0.7 | Death |

## METHODS

In this laboratory, brilliant cresyl blue, 0.3%, in alcoholic solution, is used. Slides are kept on hand which contain at one end a dried drop of dye. A drop of blood is placed on a cover slip which is inverted on the dye, and the two allowed to mix during 3-4 minutes. Under high-dry or oil, those cells which are seen to contain within their cytoplasm granules, rods, or networks are called "reticulated" and their number counted. It is customary to count 1000 red cells, in fields of about 50, and to enumerate




the number of reticulated cells seen during the counting process. The field may easily be cut down by the simple device of inserting a suitably perforated paper above the diaphragm of the eye-piece.

This is undoubtedly the simplest method for the enumeration of the reticulated cells. However, it is open to criticism in that the dye, or the cells, or both may not be evenly spread; that some of the reticulated cells might wander to the edges; that the dye might not penetrate a thick specimen. These theoretical difficulties have been met in some laboratories by finer, though more time-consuming methods. Krumbhaar[10] uses 0.3% brilliant cresyl blue in normal salt solution, and mixes the blood with the stain in a white-counting pipette, proceeding with the count as above. Instead of this, several authors mix a drop of blood with a drop of the dye in solution. Luzzato and Ravenna[11] recommend a very accurate method, that of counting the cells in a Thoma-Zeiss chamber. A red-counting pipette is used, in which blood is drawn to the "1" mark and diluted to 101 with Hayem's solution. A rather thin cover glass, which has been lightly smeared with brilliant cresyl blue solution, is placed on the counting chamber and a drop of the diluted blood is introduced. The small amount of dye present on the cover slip is sufficient to stain the red cells and thus make possible a count of the *absolute* numbers of reticulated cells, as well as of all the red cells. The above mentioned authors made numerous counts and found that there was very little, if any difference in considering the reticulated cells from their relative or absolute standpoint. This method is undoubtedly the most accurate but in checking it up with the "dry-drop" method, I have found no material difference.

Permanent preparations showing reticulated cells can be made by the combined use of the vital stain and Wright's stain following the method first used by Hawes[11a]. A drop of blood is allowed to mix with a bit of stain and is then spread out evenly. After drying, the preparation is stained by Wright's in the ordinary fashion. Thus, in same slide, one can see reticulation, nucleation, and stippling.

## ANALYSIS OF CASES

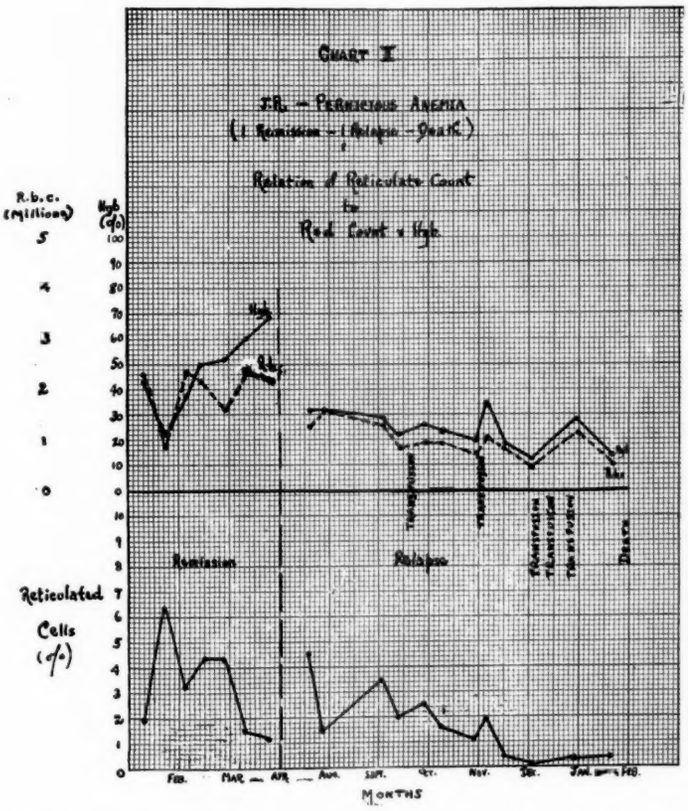
Many thousands of counts on hundreds of patients have been made in this laboratory during the past five years. However, many or most of these were done on "consultation" cases referred to the laboratory for blood work and only a single examination was made. These cases have been excluded, as having but little significance. It has been my practice in this paper to study mainly those cases ordinarily known as "blood cases", in which numerous counts have been made over long intervals.

*Pernicious Anemia.* Pernicious anemia is an ideal disease to study blood changes with corresponding variations in the reticulate count. In no other disease is it possible to see under one's eyes over long periods of time the gradual fall and rise in haemoglobin and red cells dependent (at least partially) on bone-marrow activity. In no other disease can one study the red cells during regeneration, normal periods and aplasia. It is indeed a rare opportunity to observe what happens to a single element such as reticulation or polychromatophilia during different periods in the same person's blood. During a remission, the blood counts go rapidly up, due to a bone-marrow which is regenerating so rapidly that for the nonce the forces of destruction are overwhelmed. The bone-marrow, having been stimulated and done its duty, becomes then relatively dormant and allows the forces of destruction to gain the ascendancy.

Arguing purely on theoretical grounds, then, the reticulated cells should (if they represent the younger cells of the blood) be increased before or during a remission and decreased before or during a relapse.

Little has been written of the relationship of high or low reticulate counts to the remission and relapse. Most authors are content with saying that high figures are common in the disease. Naegeli[3], in his remarkable book on the blood, states that the appearance of reticulated cells is the surest and earliest sign of a reacting bone-marrow. Drinker[12], in Oxford medicine, says that the reticulate count is a valuable and accurate index of blood regeneration. This view is held too by Christian[13]. Krumbhaar[10] states that in pernicious anemia, in which there is usually a count of from 2-5%, the count may go to 10-15% before a remission and then fall to normal with regression. Vogel and McCurdy[14] give the only case reports I have been able to find, and from six cases of pernicious anemia conclude that the reticulated cells, if high, show a well-functioning bone-marrow; if low, give evidence of aplasia. Césaris-Demel was well aware of this fact since his first work on the subject in 1899-1900[15].

How the theoretical principle enunciated above works out in practice is best exemplified in the following figures:—in 50 cases of pernicious anemia (with about 100 remissions and relapses), the reticulate count rose (in 80% of the cases) to a significant figure before a remission, and fell to or remained at a low figure (in 96% of the cases) during a relapse. By significant figure, I mean in this disease a percentage of at least 5%, preferably at least 6%. In pernicious anemia, the reticulate count is apt to be higher and more subject to variations than in normal blood. Counts up to 5% should not be be considered as presaging a remission, for evidently the bone-marrow in this disease is continually regenerating as well as continually being destroyed. It would seem that about 6% or over reticulation is needed before the destructive forces are outbalanced and remission occurs. Substantially all the 50 cases conformed to type: from a few days to a few weeks before a remission, the reticulate percentage rose to above 6, followed by a definite rise in hemoglobin and red cells, and then a fall in reticulate count; during a relapse or as a forecast of death, the reticulate count remained at a low figure—0.5—1%




The relation of the reticulate count to transfusion is an interesting one. In practically all cases, there was some rise, however slight, following transfusion. Not uncommonly, when remission was unlikely because of low reticulate count, transfusion caused apparently the necessary stimulating effect and the reticulate count jumped to a high figure and remission occurred (see cases 2, 18, 20, 35). It is rather amusing in the occasional case to see the retic-

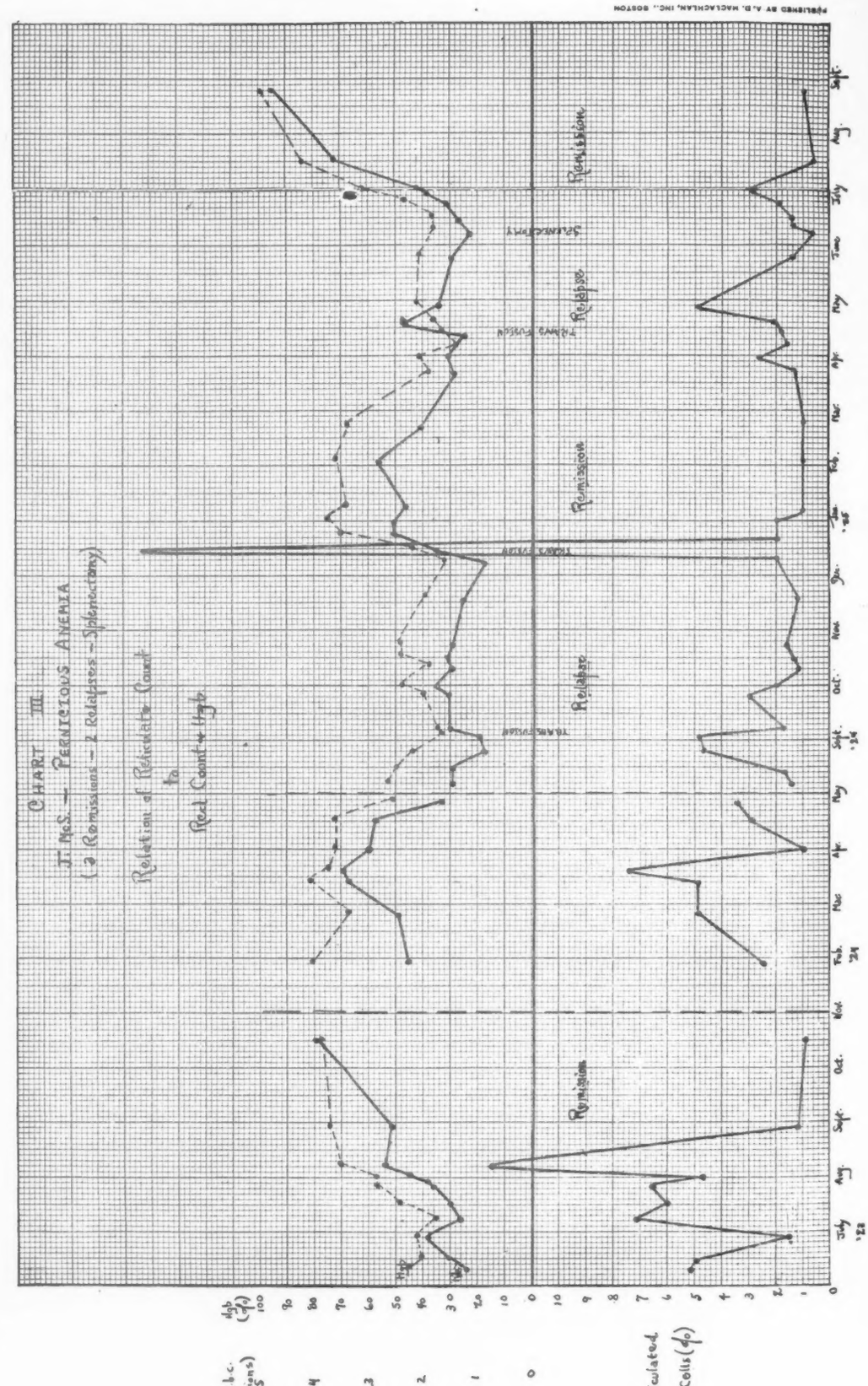


CHART III
J. Mc S. — PERNICIOUS ANEMIA
(3 Remissions — 2 Relapses — Splenectomy)
Relation of Reticulocyte Count
to
Red Count + Hgb
Remission
Relapse
Remission
Relapse
Remission
Reticulated Cells (%)
R.b.c. (Millions)
Hgb (%)

ulate count rise sharply, presaging a remission, and then because clinically the patient seemed in jeopardy, transfusion is done. Remission, which naturally occurs, is ascribed to the transfusion, when it undoubtedly would have come about without it. The futility of transfusions in the face of the continued very low reticulate counts stands out sharply as the cases are observed in their entirety.

Splenectomy in pernicious anemia—a procedure which was done on three cases in this series—failed to produce the characteristic rise in reticulate count with remission. In each case, there was complete remission, but only normal or slightly elevated reticulate counts. Whether this means that the bone-marrow is not stimulated and the rise is due simply to withdrawal of the inhibiting or destructive powers of the spleen is problematic. At any rate, the fact remains.

Table I gives a list of the cases observed and a condensed summary of their behavior with especial relation to the reticulate count. It may be of interest also to submit a few charts showing the course of the counts in detail.

It seems to me that the foregoing analyses show quite conclusively that the reticulate count is an accurate index of the function of the bone-marrow at least in regard to the red cells. Though the problem of the essential nature of the reticulated cell be unsolved; though it be still a question as to whether reticulation is performed in the cell or merely a reaction of the cell with the stain; it can nevertheless be unquestioned that here is statistical proof that a high reticulate count means active regeneration of the bone-marrow—a low count hypofunction or aplasia of that structure.

Further than that, the reticulate count, above all other signs, is the best single point in estimating accurately the amount of bone-marrow regeneration. Let us take the other undoubted signs of regeneration one by one and compare them with the reticulate count. *Nucleated red cells* are of course young reds. Their appearance in the peripheral blood stream is abnormal and bespeaks a bone-marrow irritation. Their presence in pernicious anemia is characteristic of the disease, yet can one say the same of their number? Some cases seem to have a natural tendency to show nucleated cells and to exhibit them both during the relapse and remission. Other cases, on the other hand, whether remitting or not, rarely show nucleated cells. These, though numerous in a given case, may go hand in hand with a very active destruction, and their presence, though startling to the eye, is nevertheless of but little practical significance. Take a case like that of W.F., who on a certain date shows 2% reticulated cells, but over 1% nucleated cells (2 for every white corpuscle). His hemoglobin and red count falls, but when, later, the reticulate count goes to 8.3%, and the nucleated red count is still 1%, the signal for regeneration has been given and up go the hemoglobin and red counts. These cases are repeated time and again. The nucleated red cells may represent bone-marrow regeneration but their number is by no means an index as to whether a remission will occur or not. Contrast this with the accurate and extremely delicate shifts in the reticulate count.

*Polychromatophilia* is another universally conceded sign of regeneration. Normally, one sees in the blood smears stained with Wright's an occasional off-colored, slate-gray or bluish cell. They vary in color from a grayish to a deep blue. Their number is normally extremely low—about 2 per 1000 cells over even less. In pernicious anemia, their number is often increased, especially when the blood is on the upgrade, but in the case cited above, there was only 1% polychromatophilic cells with a reticulate count which varied from 2 to 8.3%. Nevertheless, to count the number of off-colored cells per field is often of valuable assistance in estimating regeneration. Often, however, it is difficult to decide whether a given cell is polychromatophilic or not. Again, the reticulate count is a much more delicate index of regeneration. Nevertheless, it cannot be denied that the polychromatophilia of the fixed preparation runs parallel (at times) to the reticulation of the unfixed smear.

*Basic Stippling.* Although this is a sign of regeneration, it by no means follows that it is normal regeneration. Stippling is seen in various anemias and occasionally in lead poisoning, but in no way can it be correlated to the amount of active regeneration. Attempts to do so have been quite unsuccessful.

*Aplastic Anemia.*

From a consideration of pernicious anemia, which, as has been seen, shows phases of regeneration and aplasia, we pass to a disease which at no time in its course shows evidences of regeneration—aplastic anemia. This disease, extremely rare, represents a rapid failure in bone-marrow growth, with resultant anemia and death. All the elements of the bone-marrow rapidly disappear from the blood-stream—the white cells, platelets, and reds. In such a disease, one would expect the reticulate count to be almost nil and to remain constantly so until

TABLE II

APLASTIC ANEMIA—RETICULATE COUNT

| Case | Initials | Sex | Age | Reticulate Count | Hgb | Red Count | Result |
|---|---|---|---|---|---|---|---|
| 1 | R. B. | ♀ | 27 | 1.0 | 28-45 | 1.8-2.0 | Death |
| 2 | A. P. | ♀ | 43 | 0.0-2.0 | 21-12 | 0.9-0.8 | Death |
| 3 | C. H. | ♀ | 32 | 4.7-0.7 | 16-9 | 0.5-1.1 | Death |
| 4 | C. McS. | ♀ | 19 | 4.0-1.4 | 12-23 | 0.4-1.0 | Death |

death. This is borne out in the few cases that have been observed in this clinic.

*Purpura Hemorrhagica*

This disease, perhaps closely allied to the one just mentioned, represents a failure in platelet growth, resulting, accordingly, in hemorrhages and anemia. Whether or not platelet formation and platelet aplasia are closely related to red cell growth, there is no doubt but that with continued low reticulate count, the patients continue to bleed and die, but with a shift to a sustained high count, the platelets increase in number and the patient recovers. We have had in this clinic recently, however, a notable exception to this statement. (J. L.)

*The Anemias of Pregnancy.* This is a subject in itself, since during and following pregnancy there may occur secondary, primary, and splenic types of anemia. In the primary type, in which the blood goes rapidly down and has a high color index, the reticulate count behaves exactly as in pernicious anemia—a low count during the aplastic stage and a marked increase presaging a rise in red cells and recovery. The secondary types are in no wise different than secondary anemias in other conditions. I wish to present here a case which has already been reported by Dr. Larrabee[16]—one of unclassified anemia associated with pregnancy. The appended chart shows how in spite of repeated transfusion, the blood kept falling, until finally

TABLE III

PURPURA HEMORRHAGICA—RETICULATE COUNT

| Case | Initials | Sex | Age | Reticulate Count | Platelets (Thousands) | Hgb | Red Count | Result |
|---|---|---|---|---|---|---|---|---|
| 1 | F. C. | ♂ | 62 | 18-9 | 44-94 | 58-90 | 2.5-5.3 | Recovered |
| 2 | O. D. | ♂ | 16 | 2-5.8 | 60-100 | 50-70 | 2.8-4.6 | Recovered |
| 3 | A. M. | ♀ | 17 | 10.8-2.0 | 58-122 | 43-28 | 3.0-1.9 | Died |
| 4 | M. S. | ♀ | 35 | 5-13 | 42-250 | 70-90 | 3.0-4.7 | Recovered |
| 5 | L. C. | ♂ | 16 | 3-9 | 70-348 | 59-72 | 2.3-5.0 | Recovered |
| 6 | M. G. | ♀ | 17 | 9-21 | 20-136 | 25-21 | 2.4-0.9 | Death (Peritonitis) |
| 7 | J. L. | ♀ | 40 | 11-41 | 68-48 | 54-22 | 4.5-1.5 | Death |

*Congenital Hemolytic Anemia*

It is in this disease that the reticulate count assumes more than a prognostic significance, for here are recorded the highest obtainable counts. Here where continued slight bone-marrow destruction results evidently in bone-marrow irritation and unconstrained regeneration, the reticulate count is extremely high. This was first observed by the Frenchmen, Chauffard and Fiessinger[7] and corroborated by Widal *et al*[6]. Counts up to 50% are not at all rare, and it is unusual to get a percentage of less than

TABLE IV

CONGENITAL HEMOLYTIC ANEMIA—RETICULATE COUNT

| Case | Initials | Sex | Age | Reticulate Count | Hgb | Red Count |
|---|---|---|---|---|---|---|
| 1 | B. M. | ♀ | 2 | 25 | 71 | 4.9 |
| 2 | E. N. | ♀ | 7 | 9 | 89 | 4.7 |
| 3 | R. N. | ♂ | 40 | 11.2 | 80 | 5.2 |
| 4 | M. T. | ♀ | 18 | 33.6 | 81 | 4.1 |

15. This has quite a diagnostic significance. A tumor in the left upper quadrant with high reticulate count can be nothing else but spleen. Furthermore a palpable spleen with a high count equals either hemolytic or pernicious anemia, whereas a spleen with a normal or slightly raised count belongs to the Bandi or cirrhosis group. This is of practical significance in the question of operative treatment. I will cite the cases in Table IV.

splenectomy was done. The reticulate count jumped then to 7.2%, following which after another small fall (associated with a fall in reticulate count), there was rapid regeneration with reticulosis until the patient became polycythemic. This is a good illustration of the behavior of the reticulate count with regeneration —rise antedating it, high counts during its height, and gradual fall to normal counts with rise of red count to its peak.

*Secondary Anemias.* Most of the observations which have been made in this laboratory on anemias secondary to other causes, have been isolated ones, that is numerous counts during their progress have not been made. Of the dozen or more post-hemorrhagic cases followed over a fairly long period, none failed to show the characteristic course of the reticulated count with regeneration such as has repeatedly been emphasized above.

In the *leukemias* and *diseases* of *lymphoid origin,* the reticulate count may be slightly elevated, but without relation to the course or scope of the disease.

SUMMARY

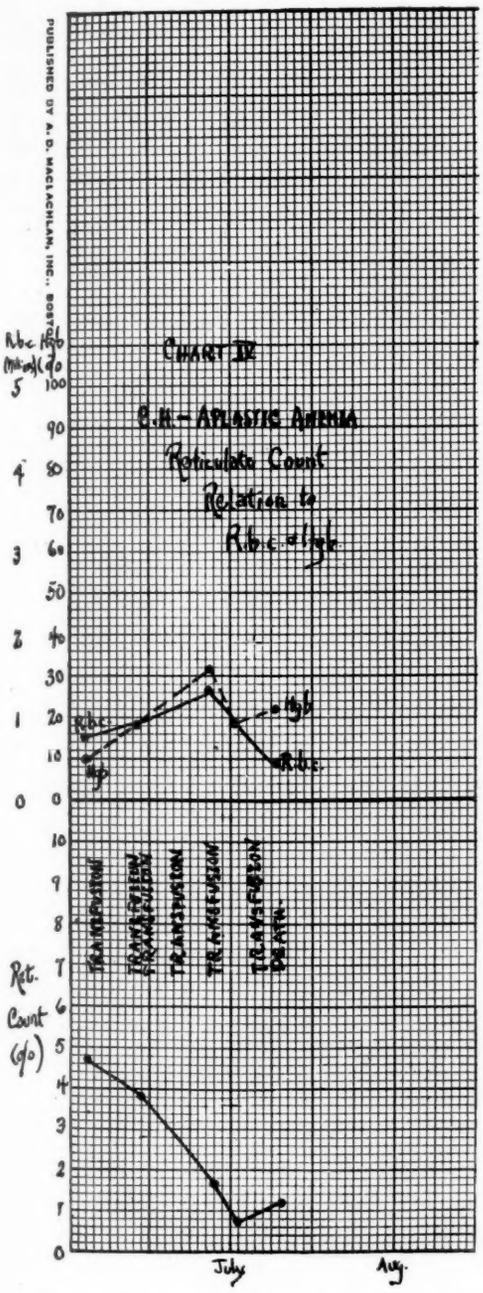
1. Routine studies of the reticulate count were made on all the cases coming to the Blood Laboratory of the Boston City Hospital, and studied by the Blood Clinic.

2. The various methods for the study of the reticulated cells are presented.

3. In 50 cases of pernicious anemia followed over periods sufficiently long for intelligent ob-

servation, the reticulate count rose in substantially all cases to a point above 6% before a remission, and fell to normal at its apex. Relapse, usually death, followed always in the wake of a continued low count.

4. The reticulate count in its relation to the




remission and relapse of pernicious anemia, and its significance as a measure of bone-marrow activity was fully discussed.

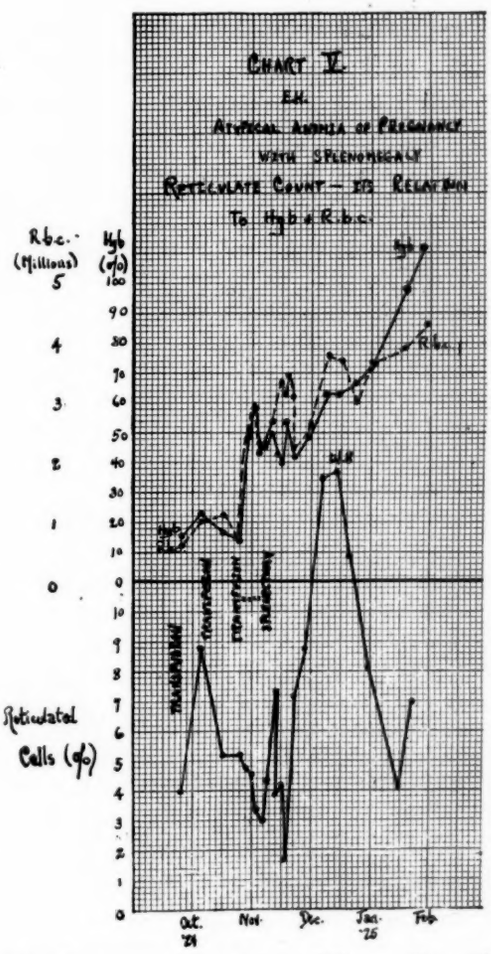
5. The various factors in the peripheral blood showing regeneration of red cells were discussed and their significance evaluated.

6. Aplastic anemia with its continued low reticulate count; purpura hemorrhagica which shows an interesting parallel between platelets and red cell regeneration; congenital hemolytic anemia with its extremely high reticulosis; the anemias of pregnancy and the secondary anemias were all discussed.

## CONCLUSIONS

1. The reticulate count of the red cells is a true index of the amount of bone-marrow activity.

a. Before and at the beginning of any remission or permanent rise in red count, the




reticulate count surpasses 6%; as the culmination of the rise is reached, it gradually falls to normal (0.5—1%).

b. With bone-marrow aplasia, the reticulate count is extremely low, practically nil.

2. The reticulate count is the *best* index to bone-marrow activity—better than nucleation of the red cells, polychromatophilia and stippling.

3. There is no better index to prognosis in pernicious anemia than the count of the reticulated cells. Although these are frequently increased, and an elevation up to 5% represents probably regeneration going on in the face of greater destruction—a sudden rise in count to above 6% presages a coming remission, over-

balancing destructive forces. Very grave are the continued low counts that bespeak forthcoming death.

4. The relapse in pernicious anemia is closely related or at least extremely similar to aplastic anemia, which with its evident bone-marrow failure, has an almost absent reticulate count.

5. Platelet regeneration and aplasia are apparently closely related to red cell growth, for in purpura hemorrhagica the reticulate count goes up before recovery and remains low with impending death from hemorrhages.

6. In the field of diagnosis, the marked reticulosis in congenital hemolytic anemia is a pathognomonic point differentiating that disease from all other anemias with large spleens.

I wish to express my appreciation and thanks to the chief of this laboratory, Dr. Ralph C. Larrabee, whose inspiration prompted this paper; and to Miss Mary F. Magee, technician, for her many courtesies.

REFERENCES

1 Ehrlich, P.: Verhandlungen d. Gesellschaft d. Charitéärzte, Dec. 9, 1880, in Berl. Klin. Wchnschr., 18:43 (Jan. 17), 1881.
2 Israel, O., and Pappenheim, A.: Ueber die Entkernung der Säugethiererythroblasten. Virch. Arch., 143:419, 1896.
2a Césaris-Demel, A.: Studien über die roten Blutkörperchen mit den Methoden der Färbung in frischem Zustande. Folia Hematol., 4, Suppl.: 1 (Oct.), 1907.
3 Naegeli, O.: Blutkrankheiten und Blutdiagnostik, 4th Ed., Berlin, 1923.
4 Pappenheim, A.: Articles in Folia Hematol.
5 Key, J. A.: Studies on erythrocytes, with special reference to reticulum, polychromatophilia and mitachondria. Arch. Int. Med., 28:511 (Nov.), 1921.
6 Widal, F.; Abrami, P., and Brulé, M.: Pluralité d'origine des ictères hémolytiques. Bull. et mem. soc. méd. d. hôp. de Paris, 24:1355 (Nov. 29), 1907.
7 Chauffard, A., and Fiessinger, N.: Ictère congénital hémolytique avec lésions globulaires. Bull. et mem. soc. méd. d. hôp. de Paris, 24:1169 (Nov. 8), 1907, and 24:1367 (Nov. 29), 1907.
8 Pepper, O. H. P., and Peet, M.: The resistance of reticulated erythrocytes. Arch. Int. Med., 12:81 (July), 1913.
9 Buckman, T. E., and MacNaugher, E.: The relative fragility of reticulated red corpuscles. J. M. Research, 44:61 (Sept.), 1923.
9a Ferrata, A.: Ueber die klinische und morphologische Bedeutung der vitalfärbaren Substanz und die basophile Punktierung der Erythrozyten. Folia Hematol., 9:253 (Apr.), 1910.
10 Krumbhaar, E. B.: Reticulosis—Increased percentage of reticulated erythrocytes in the peripheral blood. J. Lab. and Clin. Med., 8:11 (Oct.), 1922.
11 Luzzatto, A. M., and Ravenna, F.: Ueber die Zahl der vitalfärbaren roten Blutkörperchen unter normalen Verhältnissen und bei anämischen Zuständen. Folia Hematol. Archiv., 13:102 (Jan.), 1912.
11a Hawes, J. B., 2nd: A study of the reticulated red blood corpuscles by means of vital staining methods; its relation to polychromatophilia and stippling. Boston M. and S. J., 161:493 (Sept. 30), 1909.
12 Drinker, C. K.: The pathological physiology of blood cell formation and blood cell destruction. Oxford Medicine, 2:508.
13 Christian, H. A.: Article on Purpura in Oxford Medicine, 2:783.
14 Vogel, K. M., and McCurdy, V. F.: Blood transfusion and regeneration in pernicious anemia. Arch. Int. Med., 12:707 (Dec.), 1913.
15 Foà and Césaris-Demel, A.: Osservazioni sul sangue. Giornale della R. Accad. di Med. di Torino, 5:622 (Nov. 10), 1899.
16 Larrabee, R. C.: The severe anemias of pregnancy and the puerperium. Am. J. M. Sci., 92:371 (Sept.), 1925.

# A REVIEW OF OUR PRESENT KNOWLEDGE OF THE PATHOLOGY AND ROENTGEN DIAGNOSIS OF PERIAPICAL TOOTH INFECTION*

BY KURT H. THOMA, D.M.D.

THE great variation in appearance of periapical tooth infection in the roentgen picture furnishes opportunity for many differences in diagnosis and diversity of opinions. A review, therefore, of certain phases of the pathology of these infections and how they cause changes that can be recognized by the roentgen method may be found of practical value at this time.

The development of periapical infections depends on two principal factors:

(1) The nature of the local injury.
(2) The systemic defense factor.

The nature of the local injury may be mechanical (instrumentation) or chemical (irritant and escharotic drugs applied to root canals of the teeth) or infectious. Infection is generally derived from carious dentine and extended by the pulp through the apical foramen of the tooth,—in some cases the infection is carried into the periapical tissue by instrumentation. Until quite recently it was believed that the main factor governing the periapical lesions was the amount and character of the infection.

*The systemic defense factor* has only recently received proper attention through the publication of the extensive research work of Dr. Weston A. Price of Cleveland, Ohio.† He found that human beings do not react uniformly to these dental infections. Some have a great body resistance and active defense while others have a very passive defense to streptococcic infection.

The first type has a considerable immunity to the streptococcic group of diseases; the second type is highly susceptible. The personal and family history will indicate to which type the patient belongs but it must be remembered that a person who is originally non-susceptible may acquire susceptibility on account of lowered resistance from debilitating diseases or the presence of foci of infections in other parts of the body.

In the first type, the infection of the tooth produces an extensive though mild local reaction. An abscess forms showing the picture of chronic inflammation. Lymphocytes which change into plasma cells are prominent but we find also centers of suppuration where polymorphonuclear leukocytes accumulate in larger numbers. An effort is made to remove the infected tooth structure, as evidenced by erosion and the presence of osteoclasts. The lesion is called dental granuloma and replaces the bony

*From a paper read at the New England Roentgen Ray Society, January 30, 1926.

†Weston A. Price: Dental Infections, Oral and Systemic. Penton Publ. Co., Cleveland, 1925.

structure around the root apex. And it is this abscess cavity in the bone that is visualized in the roentgen picture. In this connection it should be kept in mind that the anatomical location of the abscess cavity has a great deal to do with the varying appearance in the roentgen picture. The involvement and perforation of the cortex of the maxilla or mandible gives a much more clearly outlined area on the film than results when the lesion is confined to the central part of the bone. The content of the abscess cavity has little influence on the radiability of the lesion; whether it contains a granuloma, pus or serum would not alter the picture greatly, it is the amount of bone destruction that is shown.

A word should be said about the relation of the teeth to the maxillary sinuses. In cases with a considerable thickness of bone over the roots of the teeth the same rule applies but if the superior bicuspids and molars protrude into the sinus we find that they are only covered by a thin lamina of bone forming protuberances in the floor. The amount of bone destruction is limited by these anatomical variations which must be kept in mind when interpreting roentgen pictures.

The second type of patient is either without active defence against the infection, or the defence may have become impaired or lost on account of lowered resistance of the body. Besides the various debilitating diseases, age, habits, exposure, over or under nourishment, pregnancy, grief, worry and fear have important bearing on this factor. In the second type there is only a very passive local reaction. A large granuloma is not formed and therefore there is no such walling off, no attempt to remove infected and necrotic tissue, and neutralizing of toxins as in the first group. Instead of extensive bone destruction and absorption of the tooth apex we are more likely to see proliferating changes caused by the irritating action of the bacterial toxins on the osteoblasts and cementoblasts, producing condensing ostitis and hypercementosis. The roentgen picture, therefore, will only show very small bone defects and quite often increased density of the bone and enlargement of the apex of the root.

In regard to systemic effects from these dental foci we therefore now recognize that pulpless teeth which roentgenographically show but little evidence of bone destruction at the apex may be serious sources of disease in patients who are naturally susceptible or have an acquired susceptibility to streptococcic infection. In these cases, therefore, radical dental treatment is indicated. It is necessary to extract pulpless teeth even though they show only very slight changes of the periapical bone. On the other hand this is no argument for extraction of all root-filled and pulpless teeth regardless of local and general conditions. In patients who have an active defence we often find side by side two pulpless teeth, one which shows marked change in the bone around the apex and another with practically normal periapical tissue. This can only be explained by local condition,—in one case we see the picture of a reaction which indicates the presence of infection and in the other there is no reaction because there is no cause for it. Under such condition there is no reason why pulpless teeth with normal periapical tissue may not be retained, especially if the patient is regularly seen and if the teeth are X-rayed periodically.

A periapical condition, the meaning of which is often overlooked, is that caused by an infected pulp. The tooth generally shows no indication of treatment or filling in the root canal. Clinical examination is not satisfactory. Some dentists use an electrical vitality test to diagnose the condition of the pulp and if there is a reaction to the stimulus they pronounce the tooth "vital." This, however, only indicates proper functioning of the nerves in the pulp and in fact many such teeth of which I have made sections show chronic infection and abscess formation in the tissue.* The roentgen method will not directly demonstrate pulp infection but it will show indirect evidence, such as a causative factor (caries under filling, crown, etc.) or a resulting reaction in the peridental membrane (infiltration of leukocytes and lymphocytes) with enlargement of the space between the root end and the bone. This condition should be carefully looked for when making a roentgen study of the teeth. It appears as a crescent shaped area around the apex. A pulp which is diseased is often an active focus of infection, sometimes more important than a periapical infection and not infrequently the cause of obscure neuralgia or otalgia dentalis.

In concluding I should like to point out that a roentgen examination alone is not sufficient to make a diagnosis of dental conditions. The physical condition of the patient and his susceptibility to streptococcic infection should be recognized. A very careful study is required and every case must be individually considered. While an infected tooth is a menace to health, it should be remembered that the conservation of healthy, useful teeth for proper mastication of food is of great importance, especially to the sick. Teeth, therefore, should not be sacrificed through careless diagnosis, and when certain ones must be extracted proper reconstruction of the mouth by hygienic bridges or dentures should be planned.

*Kurt H. Thoma: Pulp Disease Based on Microscopic Study. Dental Items of Interest, September, 1925, p. 637.

# NEW ENGLAND PEDIATRIC SOCIETY

## CHRONIC FATIGUE IN THE SCHOOL CHILD. A PSYCHOPHYSIOLOGIC STUDY*

BY MAX SEHAM, M.D.

*(Preliminary Report)*

After a brief address by Dr. John Lovett Morse, the newly elected president of the Society, the following paper was read:

FATIGUE is a condition most intimately related to every day life. All of us, business men, housewives, industrial workers and school children suffer from it or are affected by it at some time or other, for short periods or for long: It is a sign of normal health, yet also a symptom of disease. Varied in its manifestations it is yet simple in recognition. As common as it is and as long as we have known about it, the nature of fatigue is still obscure. Physiologists, for the last fifty years, have been studying acute neuromuscular fatigue in the laboratory; psychologists, for twenty five years past, have been investigating, by means of so called performance tests, work and fatigue curves in normal subjects; and in more recent years industrial fatigue in the worker has attracted the attention of scientists. In the school child, however, as yet no systematic study has been made of chronic fatigue. The trend of civilization towards excessive nervous and emotional habits of living affecting man already in early life makes this problem of practical and vital importance. Indeed, no problem is more closely associated with the health and happiness of the school child than the problem of chronic fatigue.

*Definition of Chronic Fatigue.* Since the term fatigue is so often misused and since there seems to exist a great difference of opinion as to just what it means let us say at once what we understand by chronic fatigue. To fatiguability, which often appears as one of many symptoms in organic disease we do not have reference. Nor do we have in mind acute fatigue which can be artificially produced in normal persons and which is the inevitable result of a definitely prescribed amount of work. Rather have we in mind a psychological syndrome, which may be primary, yet often is secondary, in which the signs and symptoms of subefficiency are usually universal, where more than one organ and more than one system is involved. The symptoms, psychic, physical or mental in character, are varied, differing at times even in one and the same individual. In some children psychic, in others physical symptoms may be predominant. The progress of chronic fatigue is usually slow and insidious. The condition may be permanent and even congenital. By that we mean that some children are born with an inferior constitution predestining them to chronic fatigue for the rest of their lives. As a clinical working definition we offer the following: Chronic fatigue is a psychophysiological syndrome of more than three months duration the state of subefficiency manifesting itself in four ways: 1. feelings of fatigue; 2. actual decrease in the individual's average mental achievement; 3. actual decrease in the individual's average physical strength and endurance; and 4. emotional unbalance.

Why is it that the child is so highly susceptible to chronic fatigue? It is because 1. he lacks adequate preparation for his school work; 2. his inhibitory and coördinating mechanism are relatively poorly developed; and 3. his emotional reactions are easily rendered abnormal.

1. *The child's lack of preparation for school work:* When, at the age of six, the child enters school, he is taken out of his natural habitat of fresh air, sunshine and freedom and almost over night subjected to an artificial regime. He is confronted with fixed responsibilities. He is required to sit still for long periods at a time. He must control his desire to whisper to his class mates or to walk about in the school room. His new environment is out of harmony with his physiological needs. He is, at that age, naturally a motor and physical animal, spontaneous and dynamic. With almost no preparation he is transplanted into a sedentary and mental life. Whereas the adult has more or less training for his work, the child has practically none to do the work required by the school curriculum. Whereas the adult has a completely developed nervous system, the nervous system of the child is in the progress of growth and strengthening. Whereas the adult can choose, at least to some extent, the kind of work he enjoys the most, and is allowed to develop his own rhythm and technic of work, the child must fit himself into the straight jacket of education, the so-called lock step system, which happens to be in vogue. We are too prone to consider school work as mere play. On the contrary, school work necessitates concentration, involves mental strain and creates nervous tension.

2. *Inhibition and Coördination:* Human efficiency depends not only on the amount of energy available for production, but also upon

*Presented at a meeting January 8, 1926.

its adequate conservation. Proper conservation of energy, on the other hand is dependent upon the development of the inhibitory and coördinating mechanism, which supervises all motor and mental actions. This mechanism protects the human machine from useless waste of energy and unrestricted activity. That this mechanism is not fully developed in the child is attested to by his impulsiveness and lack of deliberation before acting. When the child is about to start a new task he does not sit down and thoughtfully decide on the most efficient method but instead he plunges into it, trying one way and then another, until he has achieved the desired end or, as more frequently happens, he becomes discouraged and discontinues his ef-

TABLE I—Effect of Maximum Amount of Work in Foot-Pounds on Pulse and Systolic Blood Pressure. Age 12 Years.

| Subject | Age in Years | Weight in Pounds | Height in Inches | Foot-Pounds of Work | Pulse | | | | | Systolic Blood Pressure | | | | | | Remarks |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | Before Exercise | After Exercise | | | | Before Exercise | After Exercise | | | | | |
| | | | | | | Maximum Reading | Time of Maximum Reading, Sec. | Rise | Reaction Time, Sec. | | First Reading, Mm. | Time of First Reading, Sec. | Maximum Reading, Mm | Time of Maximum Reading, Sec. | Rise, Mm. | Reaction Time, Sec. | |
| J. D. | 12 | 68 | 48½ | 1 48,048 | 96 | 192 | 5 | 96 | 600+* | 113 | 140 | 5 | 158 | 40 | 45 | 570 | *Pulse 114 at 600″ |
| | | | | 2 40,275 | 84 | 168 | 5 | 84 | 600+* | 113 | 148 | 5 | 158 | 20 | 45 | 510 | *Pulse 108 at 600″ †Presence of principal might have delayed drop of blood pres. |
| I. R. | 12 | 75 | 56½ | 2 56,881 | 90 | 216 | 5 | 126 | 600+* | 103 | 138 | 5 | 141 | 50 | 41 | 420 | *Pulse 120 at 600″ |
| | | | | 1 40,481 | 90 | 192 | 5 | 102 | 420+* | 101 | 138 | 5 | 155 | 30 | 54 | 390 | *Pulse 120 at 420″ |
| A. M. | 12 | 109 | 61½ | 3 44,837 | 96 | 180 | 5 | 84 | 600+* | 136 | 188 | 5 | 188 | 5 | 52 | 330 | *Pulse 120 at 600″ |
| | | | | 1 37,740 | 72 | 150 | 5 | 108 | 450+* | 129 | 135 | 5 | 155 | 30 | 27 | 2101 | *Pulse 126 at 450″ †Bl. Pr. below normal at 240″, 116 at 450″ |
| | | | | 2 26,240 | 90 | 156 | 10 | 66 | 900+* | 132 | 169 | 20 | 169 | 20 | 37 | 150 | *Pulse 120 at 900″, 90 at 30 minutes |
| D. G. | 12 | 100 | 58¾ | 1 44,514 | 115 | 180 | 5 | 65 | 420+* | 115 | 136 | 5 | 138 | 30 | 23 | 210 | *Pulse 132 at 420″ |
| | | | | 2 30,139 | 112 | 168 | 5 | 56 | 540+* | 112 | 130 | 5 | 132 | 5 | 20 | 210 | *Pulse 115 at 540″; murmur |
| J. T. | 12 | 76 | 55½ | 1 30,709 | 81 | ... | ... | ... | 360+* | 102 | 124 | 5 | 124 | 5 | 22 | 180 | *Pulse 126 at 360″; Exp. 1 against 1½ pounds |
| | | | | 2 12,622 | 104 | ... | ... | ... | 420+* | 100 | 121 | 10 | 130 | 30 | 30 | 360 | *Pulse 120 at 420″; Exp. 2 against 1½ pounds |
| T. B. | 12 | 89 | 54¾ | 1 24,299 | 81 | 156 | 10 | 72 | 480+* | 102 | 107 | 15 | 127 | 50 | 25 | 240 | *Pulse 96 at 480″ |
| | | | | 2 29,224 | 90 | 156 | 5 | 66 | 300+* | 100 | 115 | 5 | 134 | 20 | 34 | 300 | *Pulse 102 at 300″ |
| J. B. | 12 | 88.5 | 58 | 2 28,711 | 66 | ... | ... | ... | 600+* | 98 | 129 | 5 | 136 | 20 | 43 | 360 | *Pulse 82 at 600″ |
| | | | | 1 14,856 | 60 | ... | ... | ... | 600+* | 99 | 124 | 10 | 132 | 20 | 33 | 270 | *Pulse 78 at 600″ |
| M. S. | 12 | *81.5 | ... | 25,452 | 87 | ... | ... | ... | 600+* | 97 | 129 | 10 | 133 | 50 | 36 | 450 | *Pulse 120 at 600″ |
| A. J. | 12 | 74 | 57¾ | 1 22,070 | 84 | 156 | 5 | 72 | 600+* | 114 | 152 | 5 | 152 | 5 | 37 | 270 | *Pulse 96 at 600″; murmur |
| B. B. | 12 | 62.5 | 52¾ | 2 20,853 | 66 | 132 | 5 | 66 | 540+* | 116 | 140 | 5 | 147 | 20 | 31 | 210 | *Pulse 84 at 540″ |
| | | | | 2 18,407 | 84 | 156 | 10 | 72 | 540+* | 109 | 155 | 5 | 159 | 10 | 50 | 390 | *Pulse 108 at 510″ |
| A. G. | 12 | 80.5 | 60 | 1 14,069 | 96 | 180 | 5 | 84 | 540+* | 112 | 148 | 5 | 149 | 5 | 36 | 240 | *Pulse 108 at 540″ |
| | | | | 1 16,904 | 84 | 168 | 30 | 84 | 210+* | 107 | 134 | 30 | 140 | 60 | 33 | 240+† | *Pulse 105 at 240″ †Bl. Pr. 114 at 240″ (one of first Exp. made) |
| M. R. | 12 | 74 | 56 | 2 14,667 | 84 | 168 | 5 | 84 | 480+* | 107 | 128 | 5 | 145 | 40 | 38 | 270 | *Pulse 96 at 450″ |
| | | | | 16,790 | 108 | 132 | 30 | 24 | 240 | 108 | 152 | 30 | 152 | 30 | 44 | 450 | |
| 12 cases, 23 determinations | 12 | 84.1 | 56 | 29,216 | 87 | 171.8 | 5 | 81.6 | 600+ | 110 | 136.8 | 5 | 144.2 | 24.3 | 36.2 | 323 | M. R. excluded |

forts. Undoubtedly, the inhibitory and coördinating mechanism plays a great role in the postponement as well as in the production of chronic fatigue.

3. *Emotional Reactions of The Child:* The more one studies chronic fatigue in the school child the more clearly motivation stands out as a causative influence. If the child were living in an atmosphere free from emotional stimulation the problem of chronic fatigue would be quite simple. But where is there a child today that, at one time or another, is not exposed to emotional strain, fear, worry, anxiety, excitement and nervous tensions? The child's threshold of emotional stability is on a lower level than that of the adult. He is more easily upset and incidents that are made light of by the adult often make a deep impression upon the child.

These, then are three of the most important factors which account for the high susceptibility of the child to chronic fatigue.

The experimental work that follows was carried out to establish: (1) Whether objective tests are of use in the diagnosis of chronic subefficiency of chronic fatigue and (2) whether the answers to the questionnaires by the child, the parent, the class room teacher and the gymnasium teacher give information sufficiently reliable to warrant their use in functional diagnosis.

1. *Objective tests.* It would, of course, be of great practical importance if the degree or the amount of fatigue could be measured objectively. The large number of psycho-physiologic tests that in the past have been used can be divided into performance and non-performance tests.

In a previous study of the physical capacity of normal children by means of several physical tests we came to the conclusion that no one test is a reliable index of fatigue. An analysis of Table I* which records the physical capacity determined by means of the stationary

TABLE II

COMPARISON OF ANATOMICAL MEASUREMENTS AND OF OBJECTIVE TESTS OF NORMAL AND ABNORMAL SUBJECTS

| Measurements and Tests | | Normal | | | Abnormal | | |
|---|---|---|---|---|---|---|---|
| | | Average | Med.dev. | % Med.dev. | Average | Med.dev. | % Med.dev. |
| Height (inches) | | 57.5 | 1.83 | 3.18 | 59.17 | 2.37 | 4.01 |
| Weight (pounds) | | 86.2 | 3.95 | 4.65 | 81.4 | 7.98 | 9.8 |
| Circumference of head (cm.) | | 53.13 | 1.25 | 2.36 | 53.33 | 0.87 | 1.63 |
| Circumference of chest (cm.) | | 67.4 | 3.57 | 5.29 | 61.7 | 4.58 | 7.42 |
| Circumference of biceps (cm.) | | 22.1 | 1.04 | 4.7 | 20.1 | 1.39 | 6.91 |
| Circumference of elbow (cm.) | | 23.1 | 1.41 | 6.104 | 22.3 | 1.38 | 6.19 |
| Slow movement | a | 1.51 | 0.67 | 44.66 | 1.77 | 1.08 | 61.00 |
| | b | 0.81 | 0.394 | 48.64 | 0.36 | 0.74 | 205.5 |
| Letter cancellation | a | 40.9 | 4.03 | 9.85 | 37 | 5.57 | 15.06 |
| | b | 81.7 | 11.31 | 13.84 | 75.7 | 13.04 | 17.22 |
| Steadiness test 4 hole | a | 4.13 | 3 | 75 | 8.7 | 4.88 | 50.3 |
| | b | 5.4 | 3.9 | 73.7 | 8.16 | 5.95 | 74.37 |
| 5 hole | a | 10 | 5.66 | 56 | 16.3 | 6.80 | 42.8 |
| | b | 10.8 | 6.22 | 57.6 | 15.48 | 7.21 | 46.53 |
| 6 hole | a | 20.80 | 8.00 | 38.09 | 19.30 | 9.37 | 51.22 |
| | b | 19.42 | 7.89 | 41.52 | 15.2 | 6.93 | 45.59 |
| 7 hole | a | 25 | 10.2 | 40.8 | 30.15 | 7.75 | 28.75 |
| | b | 24.4 | 11.3 | 46.3 | 31.10 | 8.63 | 27.83 |
| 8 hole | a | 32.3 | 11.59 | 35.89 | 38.85 | 8.3 | 21.29 |
| | b | 35 | 12.19 | 37.68 | 38.58 | 7.89 | 20.23 |
| Body balance (seconds) | | 118.6 | 16.31 | 13.82 | 146 | 72.39 | 56.3 |
| Stick balance (seconds) | | 14.8 | 15.9 | 10.6 | 6 | 3.51 | 58.5 |
| Tapping | a | 69.5 | 11 | 15.83 | 67.13 | 12.39 | 18.49 |
| | b | 82.1 | 7.86 | 9.59 | 75.4 | 9.57 | 12.69 |

*Taken from the American Journal of Diseases of Children, January, 1923, Vol. 25, pp. 1-45.

bicycle, shows how difficult it is to establish correct standards of physical capacity of normal children, to be used as a basis of comparison for the capacity of fatigued children. For instance, the same individual* (H.R.) will perform, at various sittings, from 40.000 to 56.000 footpounds. With so great a difference, with so high a coefficient of variability existing in normal children, the amount and degree of fatigue cannot possibly be measured accurately.

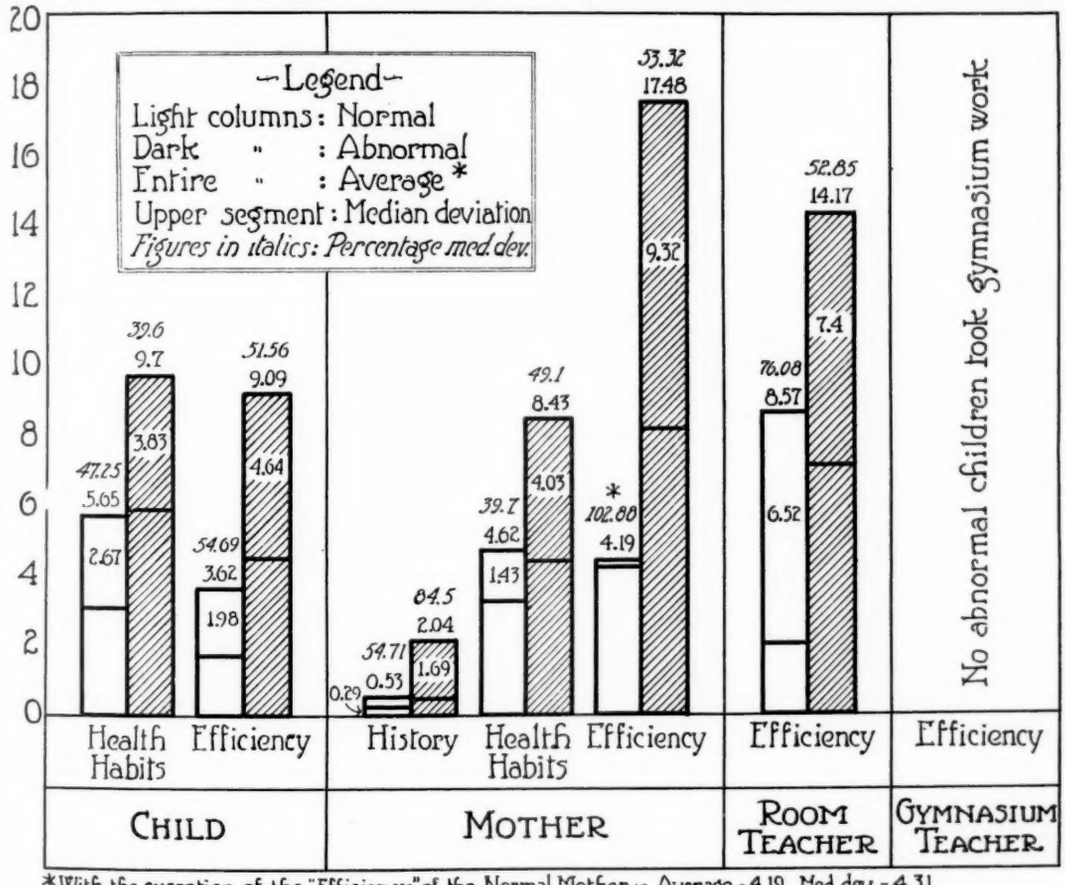
The main reason, however, for the unreliability for diagnosis of such, or similar methods must be sought in the fact that they measure solely the amount of work accomplished by one or several groups of muscles or one or several organs of the body. In order to determine the general state of efficiency or the total functional capacity of the individual, movitation must alse be measured, and that is well nigh impossible.

In Table II are recorded the results (by average, medium deviations and percentage deviation) of a study of (1) normal children used for control and (2) chronic subefficient children by means of performance and non-performance tests. On the assumption that the abnormal group might have less reserve energy than the normal group the time of performance was extended to twice that ordinarily precribed by psychologists in their measurements of acute fatigue, artificially produced.

The letter cancellation test was continued for two instead of one minute, the slow movement test was repeated 8 instead of 4 times, the tapping test 10 instead of 5 times, and the steadiness test 6 instead of 3 times. In the letter cancellation test, the child is required to check the A's on a printed sheet of letters, as speedily as possible, the number of checked A's being counted at the end of one and two minutes. In the slow movement test the child traces the circles printed on the chart as slowly as possible 8 consecutive times, in periods of two minutes each, thirty seconds being allowed for rest between two trials. In the tapping test the child makes pencil dotes (printed squares being used to facilitate counting) at the fastest rate possible. Ten trials, of 15 seconds each, with a 15 second rest period between trials, are allowed. In the steadiness test, a metal pointer is inserted by the child, into six successively smaller holes on a metal block, the child being instructed not to touch the edges of the holes. Every time the child touches the edge with the pointer the electrical current is closed and the counter

TABLE III

COMPARISON OF QUESTIONNAIRES OF NORMAL AND ABNORMAL SUBJECTS




*With the exception of the "Efficiency" of the Normal Mother.— Average = 4.19, Med.dev. = 4.31

registers one point. In the body balance test the child stands with the ball of the right foot on a wooden block. The child is urged to hold his balance as long as possible. The stick balance test requires the balancing of a yard stick on the tip of the index finger.

Letter "a" in Table II refers to the first half and letter "b" to the second half of an experiment. It is in the second half that quantitative differences between normal and abnormal children might theoretically be expected. Contrarily, a comparison of the averages in the slow movement test, shows a greater steadiness in the abnormal cases (0.36 of a circle against 0.81). This test must, therefore, be considered of no value in the measurement of chronic fatigue. The same consideration applies to the letter cancellation test, since the difference (75.7 as against 81.7) is too small to be considered sufficiently significant for diagnostic use. Also the tapping test is apparently negative, the difference in the averages (75.4 as against 82.2) certainly lies within the error of experimentation. Nor do we find that the body balance test and the stick balance test can be used for practical purposes. In the steadiness test, however, there seems to be an indication that subefficient children are less steady. In both the first and second half of the experiment there is, with the exception of the fifth hole, a consistent difference in the number of "touches" of the normal and abnormal children. Whether this test can be used for diagnostic purposes, remains to be shown by further experimentation.

TABLE IV

CLASSIFICATION OF CHRONIC FATIGUE IN THE SCHOOL CHILD, FROM A PSYCHO-PHYSIOLOGICAL POINT OF VIEW

Congenital.

1. Congenital Asthenia Universalis.
   Synonyms.
   (1) Congenital Asthenia.
   (2) Stiller's Disease.
   (3) Neuro-circulatory Asthenia.
   (4) Effort Syndrome.
   (5) Status-Thymico-Lymphaticus.
2. Neuropathic Diathesis.
   Synonyms.
   (1) Neurosis.
   (2) Neuresthenia.
   (3) Nerve Overstrain.
   (4) Hyperkenesis.

Acquired.

1. Normal physical development with subefficiency.
   (1) Psycho-physiologic.
   (2) Physio-psychologic.
2. Linear type-Asthenia.

A few words about the results of the anatomical measurements may not be out of place at this time. A comparison of the averages of the various measurements (Table II), shows that subefficient children are inferior to normal children. We shall not enter into a more detailed discussion, in this preliminary report, since a more complete study of anatomical measurements will be forthcoming in the near future.

II. *The Use of Questionnaires in Functional Diagnosis:* Our questionnaires, which are too long to be published in detail, were aimed at the general efficiency of the child as well as his health habits. The questionnaires were answered by the mother, the school teacher, the gymnasium teacher and the child himself. Table III is an analysis of the questionnaires.

TABLE V

PREVENTION OF CHRONIC FATIGUE

(1) Knowledge of family history.
(2) Recognition of inferior types as Neuropathy-Asthenia, etc.
(3) Establishment of adequate physical and mental health habits in infancy.
(4) Physical exercises (especially corrective) in infancy and pre-school age.
(5) Recognize all causes responsible for chronic fatigue.
(6) Improve general health.
(7) Prevent repeated attacks of acute fatigue due to
   1. Emotional overstrain,
   2. Organic disease,
   3. Inadequate health habits.
(8) Equalize the physiological and chronological ages.
(9) Public School System.
   1. Substitute the dynamic for the static.
   2. Substitute the individual for the lock-step.
   3. More special classes.
   4. Attention to health of teacher.
   5. Improved room facilities.
   6. Vocational selection and guidance.
   7. Conservation of special senses, ears, eyes, feet.
   8. Education in technic of mental work (rhythm, rate, use of instruments).
   9. Recesses and rests in school.
   10. Supply necessary motive and interest, based upon laws of growth and development.
   11. Decrease nervous tensions.
   12. Physical education for all children every day.

For each child, the number of answers denoting inadequate health habits or signs of subefficiency were added up. A glance at Table III shows the marked contrast in the number of positive answers between the normal and abnormal children. (1) Abnormal children have poorer health habits than normal children (9.6 against 5.65). (2) Their efficiency is almost 1/3 of that of normal children (9.9 against 3.62). (3) The figures 2.4 and 0.53 respectively indicate that their family history is more positive. (4) Their efficiency, judged by the mother is worse than that of normal children (8.43 against 4.19). (5) Their efficiency as observed by the teacher is inferior to that of normal children (17.48 as against 8.57).

*Classification of Chronic Subefficiency in the School Child:* From a Psycho-Physiological Point of View. The subefficient children whom we studied can be divided into two groups, the congenital and the acquired (Table IV). In the congenital group, the two most important types are Congenital Athenia Universalis often

TABLE VI

## DAILY SCHEDULE OF RATIONAL HABITS FOR SCHOOL CHILDREN

| Age in Years | Time of Rising | Breakfast Time | Time Between Breakfast and School (minutes) | Morning Session | | Time Between School and Luncheon (minutes) | Luncheon Time | Time *1, Between Luncheon and School (minutes) | Afternoon Session | | Supper Time | Free *2 Time in minutes | Play *3 in minutes | Miscellaneous *4 Work Time | | Bed *5 Time | Hours of Sleep | Diet in Calories |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | Begins at | Ends at | | | | Begins at | Ends at | | | | Physical | Mental | | | |
| 6 | 7 a.m. | 7:45-8:05 | 40 | 8:45 | 11:30 | 30 | 12:00-12:30 | 45 | 1:15 | 3:00 | 6:00-6:30 | 180 | 180 | 0 | 0 | 6:30 | 12 | 1500-1600 |
| 7 | 7 a.m. | 7:45-8:05 | 40 | 8:45 | 11:30 | 30 | 12:00-12:30 | 45 | 1:15 | 3:00 | 6:00-6:30 | 180 | 180 | 0 | 0 | 6:30 | 12 | 1600-1700 |
| 8 | 7 a.m. | 7:45-8:05 | 40 | 8:45 | 11:45 | 30 | 12:00-12:30 | 30 | 1:15 | 3:15 | 6:00-6:30 | 195 | 195 | 0 | 0 | 7:00 | 11½ | 1700-1900 |
| 9 | 7 a.m. | 7:45-8:05 | 40 | 8:45 | 11:45 | 30 | 12:15-12:45 | 30 | 1:15 | 3:15 | 6:00-6:30 | 225 | 200 | 25 | 0 | 7:30 | 11 | 1900-2100 |
| 10 | 7 a.m. | 7:45-8:05 | 40 | 8:45 | 11:45 | 30 | 12:15-12:45 | 30 | 1:15 | 3:15 | 6:00-6:30 | 225 | 165 | 30 | 30 | 7:30 | 11 | 2100-2300 |
| 11 | 7 a.m. | 7:45-8:05 | 40 | 8:45 | 12:00 | 15 | 12:15-12:45 | 30 | 1:15 | 3:15 | 6:30-7:00 | 255 | 180 | 45 | 30 | 8:00 | 10½ | 2300-2500 |
| 12 | 7 a.m. | 7:45-8:05 | 40 | 8:45 | 12:00 | 15 | 12:15-12:45 | 30 | 1:15 | 3:15 | 6:30-7:00 | 285 | 180 | 60 | 45 | 8:30 | 10 | 2700-2900 |
| 13 | 7 a.m. | 7:45-8:05 | 40 | 8:45 | 12:00 | 15 | 12:15-12:45 | 30 | 1:15 | 3:15 | 6:30-7:00 | 300 | 180 | 60 | 60 | 9:00 | 9½ | 2900-3200 |
| 14 | 7 a.m. | 7:45-8:05 | 40 | 8:45 | 12:00 | 15 | 12:15-12:45 | 30 | 1:15 | 3:15 | 6:30-7:00 | 300 | 165 | 60 | 75 | 9:00 | 9½ | 3200-3400 |
| 15 | 7 a.m. | 7:45-8:05 | 40 | 8:45 | 12:00 | 15 | 12:15-12:45 | 30 | 1:15 | 3:15 | 6:30-7:00 | 300 | 165 | 60 | 75 | 9:00 | 9½ | 3300-3900 |

Copyright 1923—Dr. Max Seham

*1—15 to 20 minutes of this time should be used as a rest period.
*2—By free time is meant the time between the end of the afternoon session and bed-time, the time spent at supper is not included.
*3—Part of the free time should be given to play, preferably out of doors.
*4—Only a small proportion of their free time should be given to work requiring responsibility. Work is divided into physical and mental. No child under 9 years should be requested to do any mental or physical work out of school
*5—30 minutes are allowed between going to bed and falling asleep.

referred to as Congenital Asthenia, Stiller's Disease, Neuro-circulatory asthenia, Effort syndrome and Status-thymico-lymphaticus. (2) Neuropathic diathesis, known as neurosis, neurasthenia, nerve overstrain and hyperkenesis.

The acquired group comprises children: (1) of normal physical development with symptoms predominantly pyscho-physiologic or physio-psychologic and, (2) those who are linear in type, simulating congenital asthenia yet whose history does not reveal any congenital or familial disorders.

*Causes of Chronic Fatigue:* Amongst the most common causes of chronic fatigue are: inadequate sleep, inadequate food, excessive social activities, excessive amount of outside work as peddling news papers, carrying sacks of coal, clerking in grocery stores and drug stores, excessive amount of house work and home study. Also such factors as poor ventilation, bad lighting, inadequate furniture, speed tests in school, insufficient rest periods, and a nervous and fatigued teacher on the one hand, and inadequate health habits and improper home hygiene, both mental and physical on the other, contribute to the production of chronic fatigue.

For the prevention of this common disorder we present in Table V, a scheme for prevention en masse and in Table VI, a schedule of rational habits for the individual child.

The schedule of rational habits here suggested is based on the school curriculm in Minneapolis public schools. Slight changes, therefore may be necessary to make the schedule applicable to children living in other cities and particularly for children living in country districts.

## SUMMARY AND CONCLUSIONS

I. Chronic fatigue, or rather chronic subefficiency may be defined as a psycho-physiological syndrome of more than three months duration, the state of subefficiency manifesting itself in four ways: by,

1. Feelings of fatigue.
2. An actual decrease in the individual's average mental achievement.
3. An actual decrease in the individual's average physical strength and endurance.
4. Emotional unbalance.

II. Neither the amount nor the degree of fatigue can be measured by objective tests.

III. A diagnosis of chronic fatigue is justified in the presence of feelings of fatigue, and one of the other three factors mentioned in the definition of chronic fatigue.

IV. The greater number of positive answers in the questionnaires of the abnormal group of children justifies the use of questionnaires in the diagnosis of chronic subefficiency.

V. A tentative classification of chronic fatigue, based upon observed clinical types is offered as a practical guide in diagnosis, and management.

VI. A program of prevention is suggested.

VII. A schedule of rational habits applicable to the individual child attending public schools is suggested.

Thanks are due at this time to Professor R. E. Scammon, J. E. Anderson, J. F. Anderson, all of the University of Minnesota, for their many practical suggestions.

We should also like to express our appreciation to Mr. W. W. Webster, Superintendent of Public Schools of Minneapolis, Dr. H. E. Harrington, Director of School Hygiene, for permission to work in the Public Schools, to Mr. Phillip Carlson, Principal of Roosevelt High School, and Miss S. Benson, Principal of the Trudeau School, for their splendid co-operation.

## DISCUSSION

DR. RICHARD M. SMITH: Dr. Seham has presented a subject which I am sure is a very interesting one to those of us who are dealing with children. We are all of us familiar with the problem, but I think very few have studied it as thoroughly as Dr. Seham has. What he has suggested will be of distinct benefit. He has emphasized the importance of recognizing these children who have a congenital inability to do an average amount of work, either mental or physical. Such children are common. The problem is to modify their routine so that they can live as nearly as possible a normal existence without fatigue.

In this connection it is important to determine the average requirements of sleep and rest for children and the ability to perform mental and physical tasks.

We shall all of us watch with interest the further developments of Dr. Seham's studies.

DR. FRITZ B. TALBOT: *The subject that Dr. Seham has brought up is the commonest diagnosis that we have in private practice. In many instances, it is the hardest thing we have to deal with.* I have been much interested in the way that Dr. Seham has classified and interpreted various phases of the subject. It seems to me that the question of sleep is the most important and is the most difficult to control. Father comes home and the baby sits up to see him. Some times an older sister is having a good time or there are visitors which prevent the younger child from going to sleep. *The daily nap is omitted far too early.* The number of hours of sleep the younger child gets is reduced and is followed by fatigue.

Another feature that Dr. Seham suggested, although he did not emphasize it in his excellent presentation, is the part that improper bodily mechanics plays in fatigue. *There is no question in my mind that it is one of the most*

*important elements in chronic fatigue and that although it can be corrected by a summer vacation or by rest, it is not always possible to send a child off on a vacation, whereas it is possible to overcome the poor body mechanics by corrective exercises.*

I think children should rest between their plays. After playing hard, they should rest three or four minutes before going on with their play. If you have ever watched young puppies play, you will have seen the young ones play hard and become all tired out and, instead of going on with their play, they lie down and rest a few minutes. After a few minutes they are refreshed and go ahead with their play once more.

DR. KENNETH D. BLACKFAN: I was very much interested in hearing what Dr. Seham had to say about the very important question of fatigue. He has added, I think, another thing for us to do in this peculiar type of child. We have been educated to correct the physical defects and the diet of the child, and now Dr. Seham has presented to us the very interesting problem of regulating the life activities of the child. He has done it in a very careful way and his talk should be of great help to us.

DR. CHURCHILL: Dr. Seham's interesting work is going to have a more widespread and far reaching effect than most of us realize.

I attended a meeting of the Juvenile Court in the West last year. There were a good many cases brought up and a considerable number of them would have fitted into Dr. Seham's abnormal class. I cannot help but feel that this sort of work is striking right at the roots of one of the most serious problems in this country. I believe that the study of such children as these with the application of Dr. Seham's methods is going to do a great deal to prevent the great numbers of children coming to the Juvenile Court.

If Dr. Seham has not been following this type of child, may I suggest that he ask the Juvenile Court to keep in close touch with him and let him know if any of these individuals reach the Juvenile Court, the abnormal ones I mean.

DR. LEWIS W. HILL: Dr. Seham's paper has struck a very popular note in our profession.

I think the question of sleep is one of the most important points in this subject. I think the daily nap is essential for these young children and yet you find very few children over four years of age taking a nap. They grow out of the habit. Take a child who has not been in the habit of taking a rest; you cannot make him take one. I think it is important to get him to take a rest after dinner. The children who go to public schools cannot do this very well if they have some distance to go.

This leads to another point to which I am greatly opposed and that is two sessions of school. I think it is one of the worst things we have to deal with. Every chance I get I take children out of school for the afternoon session. I feel it is much more important for him to have his rest after lunch and then go out in the fresh air.

DR. FREDERICKA MOORE: I was very much interested in Dr. Seham's paper. There are two points which I should have liked Dr. Seham to discuss in his paper. I wish he had said something about the mental age. Our children begin school at six years of age. Many times the child has the mentality of a four-year old or a five-year old child. Just where would you put this child in your scale?

In the table where he speaks of school recess, studies, etc., I should like more information as to the time of day this recess occurs, the hours at which studies occur and the number of home studies he should have.

DR. SEHAM—IN CLOSING: As I go on with this study, I should probably change some of the titles of my subdivisions. I feel quite sure that there is something in fatigue which we can help to avoid and that we ought to try to help these abnormal children to live a life like that of a normal child as nearly as possible.

# MEDICAL PROGRESS

## PROGRESS IN THE STUDY AND TREATMENT OF CARDIOVASCULAR DISEASE IN 1925

BY T. DUCKETT JONES*, M.D. AND HOWARD B. SPRAGUE, M.D.

CARDIOVASCULAR disease continues to hold a prominent place in medical literature. The literature is voluminous, to say the least, and though there have been no remarkable advances in the field during the past year, there have been many worth while articles and reports adding quite a bit to the store of knowledge, with an addition here and there of definite value. Some 500 papers have been included in this survey of which about 100 are omitted in the review, and though it is fairly inclusive, no attempt has been made to cover the entire field in an exhaustive manner. The outline is sim-

*Dalton Scholar, Massachusetts General Hospital, 1925-1926.

ilar to that used by one of us in a review of the literature one year ago in the BOSTON MEDICAL AND SURGICAL JOURNAL (192:799, 1925).

### I. ANATOMY AND PHYSIOLOGY

The anatomy of the cardiovascular system, as in the past year or two, has received no remarkable advancement. Congenital cardiac anomalies are reviewed elsewhere. However several anatomical papers of some note have appeared, and there have been a number of interesting physiological observations.

Master (*Jour. Lab. and Clin. Med.* 1925, 10:603) reported from a study of 13 human hearts that fat was always found microscopically in the sarcoplasm between muscle fibrils. There was no relation between the amount of fat and the state of nutrition at the time of death, age, color, or sex. Even the so-called pigment or brown atrophy of the heart was found in moderate amounts in 2 healthy boys, 8 and 15 years old.

Kerr and Mettier (*Amer. Heart Jour.* 1925, 1:96) have studied the circulation of the heart valves, using pigs' hearts, and injecting india ink through the coronaries. The valves and chorda tendinae had a fine network of vessels. They present this as further confirmation of the embolic conception of endocarditis.

DeBuys and Samuel (*Amer. Jour. Dis. Child.* 1925, 30:355) have made a number of observations on 400 subjects for 39 months, concerning the growth of the heart. They conclude that changes in the position of the apex beat from above downward and from without inward are due to a more rapid growth in the size of the thorax as compared with the growth in the size of the heart, and that there is no element of rotation in the heart, nor does it assume a more vertical position as age advances during the period studied. They found the average weight of the heart is 0.76% of body weight at birth, and 0.46% in adult life.

Géraudel (*Presse Méd.* 1925, 33:1283) gives an illustrated description of the artery to the sino-auricular node, emphasizing what he believes to be a connection between functional changes in the node and a disturbed blood supply. He thinks this is a terminal artery, without anastomoses, analagous to arteries in the brain.

"Capillaries"—Cohn and Crawford (*Jour. Clin. Invest.* 1925, 1:575) demonstrated by moving pictures capillaries in normal individuals and patients with auricular fibrillation, mitral stenosis, and aortic regurgitation, etc. Sometimes capillary pulsations were seen in persons with aortic incompetence. Callander (*Jour. Am. Med. Assoc.* 1925, 84:352) also gives a report of photomicrographic studies of morphology of surface capillaries in health and disease, including the anatomy and technique. Henderson (abstract *Jour. Am. Med. Assoc.* 1925, 84:393) believes that by great variations in the size of the capillary bed, it is possible to furnish oxygen and remove carbon dioxid in suitable quantities, and that the very important function of the capillaries in taking care of the enormously changing needs of the tissues may be shown mathematically. Wearn (*Jour. Clin. Invest.* 1925, 1:572) studied the capillaries and Thebesian vessels of human and cat hearts. India ink was injected intracardiacally in living cats and from 2000 to 3500 capillaries per square millimeter were found, going as high as 5700 after injection of histamine. A reserve supply of capillaries in the heart was thus suggested. Perfusion of the coronary arteries with saline solution was carried out and quantity of outflow from all sources determined. In perfusing the coronary sinus and veins all the perfusate escaped through the Thebesian vessels, while the perfusate through the Thebesian vessels escaped through the coronary sinus. These findings hold true in dead cats' hearts, and show the existence of a large direct connection between the coronary arteries and the chambers of the heart as well as the well known communication between the Thebesian vessels and the veins*. Brown (*Arch. Int. Med.* 1925, 35:556) made an interesting study of the skin capillaries in 8 cases of Raynaud's disease, giving the findings at various stages of the condition. In the normal compensated periods, many of the vessels disclosed evidence of disturbed capillary tone, abnormal flow or complete stasis.

Experimental Heart Disease—Hermann (*Amer. Heart Jour.* 1925, 1:213) calculated the proportional heart weights of 200 normal dogs —divided by the author's midseptal line method and by Lewis' septum separation method. The normal left ventricular to right ventricular weight ratio was found to be 1.393, with a minimum of 1.153 and a maximum of 1.773 by the author's method. The heart weight to body weight ratio was .00798 with a minimum of .00600 and a maximum .00994.

Allan (*Heart*, 1925, 12:181) presents a schema of the circulation with experiments to determine the additional load on the apparatus produced by conditions representing valvular lesions. He thinks it unwise to suggest that any mechanical schema of the circulation be applied to the human circulation, because conditions vary so, but assumes that the observations can be regarded as approximately portraying the phenomena produced by actual cardiac valves. Conclusions from his observations are very interesting: Defects in cardiac valves add a definite load to the work of the heart; Regurgitation defects add a greater mechanical load than those producing obstruction; Aortic stenosis is

*At a recent meeting of the New England Heart Association, December, 1925, Gross suggested the possibility that a considerable amount of this perfusate might leak through the vessel walls and so give rise to incorrect conclusions.

relatively benign, and uses up relatively little reserve; Mitral stenosis is relatively benign at slow rates of beating, but becomes serious as diastole is shortened at rapid rates; Aortic incompetence is mechanically the most serious of single valvular flaws; Mitral regurgitation is intermediate between aortic stenosis and aortic regurgitation; increased head of pressure readily compensates aortic stenosis or mitral stenosis, less readily mitral regurgitation, but not aortic regurgitation, even when the head of pressure is increased by 80%; increased rate with shortening of diastole, compensates the effect of aortic regurgitation and aortic stenosis, and less readily mitral regurgitation and aggravates the effect of mitral stenosis; mitral regurgitation adds a serious load when combined with aortic lesions.

Beck, Cutler, and Holman (*Jour. Clin. Invest.* 1925, 1:589) have studied the effect of interventricular fistulae produced in animals on blood pressure, blood volume, and changes in the heart size. These studies demonstrated that the capacity of the chambers of the heart and the alterations in their musculature seem to be determined by the minute volume flow through them.

Felix (*Deutsche Ztschr f. Chir.* 1925, 190:178) produced experimental valvular lesions in dogs' hearts and then tried to influence them by modifying the pericardium. He concluded that the function of the pericardium is predominantly mechanical. Making it smaller hindered the heart action, while enlarging it had a favorable influence on mitral and tricuspid valvular defects, but not on aortic or pulmonary defects, as there is no compensatory dilatation with the latter as with the former.

Sino-auricular conduction—Resnik (*Arch. Int. Med.* 1925, 36:788) reports the results of 6 experiment on dogs, obtaining electrocardiographic evidence of activity of the sino-auricular node. Sinus arrhythmia was found in all dogs. Pituitary extract was used for its vagus effect in producing block. He found no evidence whatever of any nodal activity during the so-called block, and believes the condition is due to failure of the impulse to arise in the sino-auricular node during the pause, and not to failure of conduction. He urgently suggests the term sino-auricular standstill in preference to block and draws attention to the close similarity between this condition and sinus arrhythmia. Rothberger and Scherf (*Wien. Klin. Wchnschr.* 1925, 38:1301) believe that there are two paths of conduction between the node and the auricle. Experimentally, elimination of one of them caused changes of the auricular electrocardiogram, and ligation of both isolated the sinus.

"Effect of Anoxemia on the Heart"—Resnik (*Jour. Clin. Invest.* 1925, 2:117) reports that, in the normal heart, anoxemia produces comparatively slight changes in intraventricular conduction. At first the Q-R-S interval shortens; later, it lengthens. He thinks that ventricular aberration in clinical cases is an indication of more or less serious disturbance of the function of the Purkinje tissues. Ventricular fibrillation is prone to develop during anoxemia. He also found (*Jour. Clin. Invest.* 1925, 2:125) that the sino-auricular node is highly sensitive to anoxemia. There was first a brief period during which the rate of impulse formation is accelerated, followed rapidly by progressive slowing of the intrinsic rate of the heart. Anoxemia diminishes the rate at which a circus movement travels throughout the auricle. The early effect of anoxemia, when vagal tone is removed, and the auricles stimulated by a faradic current, is to predispose to fibrillation; the late effect is to inhibit the development of fibrillation. Takeuchi (*Jour. Physiol.* 1925, 60:208) found that the reduction of oxygen in the air breathed by a cat produces immediate enlargement of the heart, and the readministration of oxygen produces immediate reduction.

"Circulation rate"—Perhaps no field in physiology at the present day has so many capable investigators carrying out studies, as the complex problem of circulatory effects of respiration, blood flow, blood volume, etc. The problems are far from being settled definitely in many fundamentals, but doubtless much of great advantage will in time come from the investigations. Henderson and Haggard (*Amer. Jour. Physiol.* 1925, 73:193) present a new method of measuring blood flow, easy both for the subject and investigator. Slighly soluble vapor of ethyl iodid is inhaled, and the comparative content in the inspired air and the arterial blood is estimated. They found that the circulation can call on three reserves when adjustment necessary for muscular work is performed to meet the increased demand for oxygen: 1. Increase of pulse rate and fullness; 2 Increase of heart stroke volume; 3. Increase of arteriovenous oxygen difference or percentage utilization. For ordinary exertion only the 1st part of the above is necessary. Thirty-five liters per minute was found to be the upper limit of blood flow. Rabinowitch (*Arch. Int. Med.* 1925, 36:239) calculated the relative output of the heart per beat from the results obtained by the direct determination of the oxygen content of the arterial and venous blood in ten normal persons and ten persons suffering from various heart lesions, associated with congestive failure. There were marked differences noted between the normal and pathologic cases in all instances, and there was some general agreement between the clinical picture and the laboratory data in the latter. Field and Bock (*Jour. Clin. Invest.* 1925, 2:67) studied

orthopnea and the effect of posture on rate of blood flow. They found the circulation rate to be much slower in the upright position than when lying down, due to gravity effect in preventing return venous flow. Relief of dyspnea they think probably due to reduction of pulmonary congestion by upright position. Goldschmidt and Light (*Amer. Jour. Physiol.* 1925, 23:173) find that at a given oxygen unsaturation, the color of the skin depends on the ratio of capillary and venule dilatation. An arm gets blue when held down, the cyanosis being unrelated to oxygen unsaturation, but produced by increased peripheral venous pressure. Field and Bock (*Jour. Clin. Invest.* 1925, 1:581) show by experiments that the main action of Adrenalin appears to be to increase blood flow by greatly increasing the output of the heart. No demonstrable peripheral vascular constriction, due to adrenalin, was noted in the experiments.

"Reactive Hyperemia"—Lewis and Grant (*Heart*, 1925, 12:73) have studied reactive hyperemia in man. They find a state of vasodilatation when the arterial supply to a limb is occluded. The degree of vasodilatation after long occlusions (10 minutes) is not dissimilar to that produced by brief exercise of the muscles included in the plethysmograth, and the durations are of the same general order. The authors do not believe that the vasodilatation is due directly or indirectly to a deficiency of pituitary hormone. Deficient blood supply is its obvious primary cause, though it is highly improbable that it is due directly to the lack of any single blood constituent, but is thought to result from the accumulation in the extravascular tissue fluids of slowly diffusible substances, the products of metabolism during the period of circulatory disturbance. The mechanism responsible is frequently or constantly in action in physiologic circumstances and has much in common with that which locally controls the tissue circulation responding to tissue injury.

"Effect of Effort on Size of Heart"—Gordon (*Amer. Jour. Roentgenol and Radium Therapy*, 1925, 14:424) made observations on animals and marathon runners. Rabbits with normal and abnormal hearts were exercised to exhaustion. Roentgenographic examination showed the hearts to be diminished in size immediately after strenuous effort. No evidence was found of dilatation. The same was true in most instances in marathon runners, immediately after the race, returning to normal size within a few hours. No evidence of any permanent enlargement in the marathon runners could be attributed to prolonged athletic activity. He found that exercise does not produce cardiac dilatation, and that prolonged athletic training for years fails to develop hypertrophy of the heart.

"Sensibility of Heart"—Waugh (*Lancet*, 1925, 2:1054) makes an interesting report of a case in which he performed an exploratory pericardiotomy with purely regional and local analgesia. A fibroplastic pericarditis binding tightly the whole heart muscle to the parietal pericardium, was found. Ten or twelve minutes was required to free the heart, the entire operation lasting about thirty-five minutes. At no stage of the operation did any stimulus produce any sensation that the patient was able to record in consciousness; nor was this the sequence of amnesia, since he can relate fully at the present day all the ordinary details of the operation which was performed five years ago.

"Asynchronism of Ventricular Contractions"—Katz (*Amer. Jour. Physiol.* 1925, 72:655) gives experimental evidence which he believes justifies the conclusion that right and left ventricular systoles and also their ejection phases neither begin nor end simultaneously, and as a rule are of unequal duration. He suggests that the T wave may be due to unequal cessation of activity in the two ventricles.

"Dynamics of Ventricular Systole"—Katz and Feil (*Heart*, 1925, 7:171) measured the duration of the systolic phases of nine cases of aortic stenosis and thirteen of aortic insufficiency, using records obtained by optical methods of registration. These were compared with normal values at corresponding heart rates. They found that the ejection phase and total systole are definitely prolonged in aortic stenosis. In aortic insufficiency, the ejection phase is lengthened, and total systole is approximately normal in duration.

"Sympathetic Paraganglion on Left Heart"—Wassermann (*Deutsche Med. Wchnschr.* 1925, 51:1855) draws attention to the chromaffine tissue discovered by Wiesel in the neighborhood of the left coronary artery, and believes that Wiesel's organ may be the origin of the reflex angina pectoris.

Rylant (*Compt. Rend. Soc. de Biol.* 1925, 93:823) claims to have removed the Keith-Flack node, and implanted it in the tissue of the right auricle in the same dog. This retarded the heart rhythm, and the point of origin of the heart impulse seemed to be shifted to the region of the coronary sinus. The implanted node grew to the auricular tissue, and seemed to control the heart automatism. He concludes that the influence of the graft on the heart must be of hormone nature, since all the nerve and muscle connections had been severed.

Hilton and Eichholtz (*Jour. Physiol.* 1925, 59:413) assert that the coronary vessels are extremely susceptible to change in the oxygen tension of the blood flowing through them, their state of contraction being almost proportional to the oxygen tension of the blood. A fall of oxygen saturation of the hemoglobin below 20% causes maximal dilatation of these vessels.

There is no evidence that any other nonvolatile metabolites are concerned in the "regulation" of the coronary flow. The increased flow in asphyxia is sufficiently accounted for by anoxemia.

Cohn (*Jour. Exper. Med.* 1925, 42:291) presents a curve showing distinctly the changes that take place in the rate of the heart beat during the development of the chick embryo.

## II. ETIOLOGY AND PATHOLOGY

"General"—a new edition, the Fourth, of Sir James MacKenzie's Diseases of the Heart (*Oxford University Press*, 1925), has appeared during the year. The proof had been read just prior to his death, and Lady MacKenzie has had it published. The edition is somewhat fuller than the previous one, changed slightly in context, but without great additions. It is, however, delightful to read, and of definite value. Crummer (*New York: Paul B. Hoeber, Inc.*, 1925) has published a book on The Clinical Features of Heart Disease. The author is quite dogmatic in many instances concerning problems far from settled, but in general is sane and satisfactory in his clinical impression of heart disease.

White and Myers (*Amer. Heart Jour.* 1925, 1:87) have somewhat revised their Classification of Heart Disease as prepared by them about four years ago. The classification is based largely on etiology, but includes also two further important factors, without which a diagnosis is incomplete, namely, the structural change or changes, and the functional ability of the heart. All three are essential.

Dublin (*N. Y. State Jour. Med.* 1925, 25:986) gives statistical aspects of 1000 cases of organic heart disease. Eighty-eight per cent showed enlargements, among whom 1 in 5 had enlargement of the heart only. Hypertrophy was the commonest impairment. Mitral insufficiency was diagnosed in approximately half of all the cases. Mitral stenosis was present in forty-four per cent; but, in nearly all of these cases mitral regurgitation was also present. Aortic insufficiency was present in about fifteen per cent; aortic stenosis in about three per cent; aortitis in seventeen per cent, and aneurysm in a little over one per cent.

Wood and Jones (*Va. Med. Monthly*, 1925, 52:8) report 150 cases of heart disease in Virginia, thirteen per cent of which were hypertensive; forty per cent arteriosclerotic; thirty-four per cent rheumatic; ten per cent syphilitic; and three per cent congenital.

Heart Disease in England and Wales has been studied at length by Young (*Lancet*, 1925, 2:590) and he found the mortality in the counties of England and Wales shows quite definitely a tendency to a special distribution in which the higher death rates are found in the counties toward the west. A wide variance from the figures of Haviland was found, data collected for an earlier epoch (1875). He concludes that the relative incidence of acute rheumatism appears to be the essential factor that determines the incidence of heart disease, although other factors, possibly mainly environmental, may have some influence on the rate of mortality in different districts.

"Congenital Heart Disease"—Emerson and Green presented a clinical study of 203 cases of malformations of the heart before the New England Heart Association, March 19, 1925. It is an interesting and valuable contribution to the knowledge of congenital heart disease. In 20 years at the Children's Hospital, Boston, 0.002 per cent of admissions had cardiac malformations, the diagnosis in most of them being difficult and obscure in view of all the defects which may be present in a single heart. Of the cases coming to autopsy only 3 out of 35 had been correctly diagnosed. Three-quarters of the severe cases died before the age of two years. There were many septal defects, especially among the survivors; pulmonic defects were more frequent among fatal cases. They found electrocardiograms useless, and the X-ray helpful in diagnosis. Signs and symptoms were very variable. The simplest method of classification is according to the degree of cyanosis following Doctor Maud Abbott's classification. Sudden death may occur. A patent ductus arteriosus was associated only with systolic (not continuous) murmur in infancy.

Holman (*Bull. Johns Hopkins Hosp.* 1925, 36:61) has given an interesting study of patent ductus arteriosus, reporting 28 cases, and dividing them into 3 types. He considers the condition as an arteriovenous or a venoarterial fistula, resulting in an abnormal routing of a part of the circulating blood. Sixteen cases fell in Type I, in which the evidence indicated that the flow occurred from pulmonary artery to aorta. The right heart showed an abnormal development, in most instances equal to, and in several cases in excess of, the left heart. A dilated tricuspid ring, a dilated pulmonary artery, and a dilated aorta beyond the entrance of the patent ductus, were recorded as characteristics of this group. Twelve of the 16 cases showed marked cyanosis (not mentioned in the other 4). In 9 cases evidence indicated that the flow of blood occurred from aorta to pulmonary artery, and are classified as Type II. These cases showed a preponderate hypertrophy of the left heart, with a dilated aorta to the point where the ductus was given off. There was cyanosis in only 2 cases of the group. Three cases were cited in which there were no cardiac changes (Type III). This may be attributed to the narrowness of the canal (diameter 3 mm. or less) allowing only a small volume of blood to be deflected through the opening. Three

cases of idiocy fell in Group II, in which, so it is assumed, there is an interference with the proper development of the brain. The same author (*Arch. Int. Med.* 1925, 36:516) studied the relationship between increased minute volume flow and cardiac enlargement in peripheral arteriovenous fistula and patent ductus arteriosus. An intimate interrelationship was demonstrated. He also studied the volume flow of blood through the heart in the presence of intracardiac fistulas (patent interventricular septum, patent foramen ovale, con triloculare, etc.). In the presence of these fistulas the volume flow of blood through the heart is unequally distributed among the several chambers, and theoretically, the developmental response in these chambers should be commensurate with the volume flow through them. There is clinical evidence in favor of this view provided by 10 cases of congenital anomalies of the heart recorded in the literature.

Reid (*Arch. Surg.* 1925, 10:601 and 2:236) discusses abnormal arteriovenous communications, acquired and congenital. In his 33 cases, six were congenital. He believes that extripation of the involved vessels yields the best results in general. Twenty-six of the 33 cases were cured by operation, 3 improved, 2 not treated, and 2 died.

Abbott (*Ann. Clin. Med.* 1925, 4:189) discusses the incidence of infective endocarditis in 555 cardiac anomalies "of clinical significance". Six cases are reviewed, four of which have not been previously reported. The conclusions drawn are that infective endocarditis is extremely common in patients with congenital cardiac defects; the process is most commonly subacute bacterial and this is especially true of anomalies in the left heart; in septal defects, and patent ductus, an acute bacterial infection is relatively more frequent and here the lesion is usually right sided and gives unmistakable evidence, both by this fact and the presence of infarcts in the pulmonary circulation and absence of these on the systemic side, that the shunt in these abnormal communications is arterial—venous under the ordinary conditions of the circulation in health.

Nasso (*Pediatria*, Naples 1925, 33:505) reports a case of fetal endocarditis.

There have been a number of interesting case reports. Lutembacher (*Presse Méd.* 1925, 33:236) reports a case of congenital mitral stenosis with interauricular opening. He explains the latter condition as due to a reflux of blood in the foramen ovale, as a result of the mitral stenosis interfering with its closing. Melle (*Med. Jour. South Africa* 1925, 20:315) gives a case in which the heart lay almost horizontally in the thorax. It was almost square; the right side was dilated and somewhat hypertrophied. The ductus arteriosus was widely patent, and the left pulmonary artery appeared to arise from it; the lumen of the ductus was equal to the lumina of the 2 pulmonary arteries. There was marked stenosis of the isthmus of the aorta, and a marked defect in the interauricular septum. Cutler and Wilens (*Amer. Jour. Dis. Child.* 1925, 30:76) report a case of ectopia cordis in which an unsuccessful attempt was made to cover the heart with skin flaps. There was also a patent foramen ovale, patent ductus arteriosus and stenosis of the aortic isthmus. Macgregor (*Edinburgh Med. Jour.* 1925, 32:251) cites a case with a large heart, in which there was absence of the right auriculoventricular orifice, defects in both the interauricular and interventricular septa, and a patent but narrow ductus arteriosus. No cardiac defect was suspected until the day of death at seven weeks of age.

"Rheumatic Heart Disease". Justly the literature on rheumatic heart disease continues to hold a prominent place. There have been several excellent general summaries of the present status of our knowledge of this important group, each adding points of value. There also seems to be an increasing doubt in the minds of many as to just how the causative agents differ in rheumatic endocarditis and subacute bacterial endocarditis, and the pendulum is apparently beginning to swing somewhat in favor of the theory that the two may be caused by the same agent and are but different degrees of the same process.

Coombs (*Rheumatic Heart Disease*, New York: William Wood & Company, 1924) has published an exhaustive study of the rheumatic infections, and the book is well worth while. It is undoubtedly too long, and there is a certain amount of repetition, but it gives admirably the signs, symptoms, pathology, progress, and treatment, from a broad clinical experience in handling these cases, and is beyond doubt an authentic work. It is interesting to note Coombs' figures, that 69% of his rheumatic patients had severely crippled rheumatic hearts, and 72.4% of these died before the age of 40 years, only 27.6% living past this age.

Clawson (*Jour. Infect. Dis.* 1925, 36:444) isolated streptococci in 20 cases of well defined acute rheumatic fever, rheumatic endocarditis or chorea. All but 7 were isolated from the blood prior to death. This streptococcus belongs to the viridans group, and does not seem to be a specific strain. By injecting these organisms in animals, lesions similar to those occurring in human rheumatic lesions can be produced. Clawson is of the opinion that rheumatic fever with its associated lesions is produced by streptococci and believes there is a strong suggestion that rheumatic endocarditis and subacute bacterial endocarditis may be produced by the same organism and are but different degrees of the same disease.

Swift, Andrews, and Derrick (*Assoc. Amer.*

*Phys.*, May, 1925) review the two theories as to the manner of development of rheumatic valvular disease: first, implantation of the causative agent at a certain point on the valve, causing necrosis of the endothelium with underlying cardiac structures; second, that the infectious agent is brought to the valve through the circulation of the valve itself. They point to the presence of Aschoff bodies throughout the myocardium in many cases of fatal rheumatic fever, the characteristics of the subcutaneous nodules, and the pathology of the joint lesions, all being interstitial in character. They feel that the rheumatic process in the valves is also interstitial in character. The inflammatory process, occurring early in the valve substance, shows that rest and prolonged treatment are very essential.

MacCallum (The Harrington Lecture, *Jour. Amer. Med. Assoc.* 1925, 84:1545) gave a complete summary of the present knowledge of rheumatic infections, with slides of microscopic sections of nodules, involved valves, Aschoff bodies, etc. He points out that there need be no apparent joint trouble, which is usually of secondary importance, and also suggests that some myocardial disability, block, etc., may be the result of compression or of thrombosis (by injury) of some of the coronary arteries. Thayer (*Bull. Johns Hopkins Hosp.* 1925, 36:99) analyzed closely 25 cases of acute rheumatic heart disease of indisputably pure rheumatic origin without demonstrable complicating bacterial septicemia. In 68% of cases, infected tonsils or pharyngeal adenoids were noted. Five had peridental infection. In 72% there was a polyarthritis during the attack, and in an equal number there was a history of preceding arthritis. All had had arthritis during the attack or previously, save one, a child of 7 with a mild acute chorea and grave myocardial changes dating from a previous attack. The onset was usually rather vague and indefinite, except those in which it began with a polyarthritis. The arthritic symptoms were slight in a large proportion of the cases, the disease being one of the heart rather than of the joints. Unaccountably persistent fever in association with chorea or mild atypical arthritis was especially significant. Renal infarctions were found post mortem in 3 cases; thrombi in the auricular appendages in 5; and pulmonary infarctions in 3. Either acute or chronic myocardial changes occurred in all of the 24 cases, in which the heart muscle was available for examination.

Bertram (*Brit. Med. Jour.* 1925, 1:496) reports a series of 141 cases of rheumatic infection observed over a period of 5 years, and followed up 2 years later. No case occurred during infancy and 60% were from 6 to 10 years of age at the onset of the rheumatism. Girls were affected more frequently than boys, more marked in the case of chorea (3:1). Arthritis was most prevalent in October and November (32%), and chorea in February and November (32%) (Glascow). The incidence of lesions considered was as follows: 76% carditis; 73% arthritis; 47% chorea; 28% tonsillitis; 7% subcutaneous nodules; 6% skin lesions. Of the total cases 20% died; 25% of the fatalities were within 3 months; 51% within one year; 75% within 2 years, from the onset of rheumatism. Pericarditis and subcutaneous nodules were the lesions indicating most severe infection. Early hospital treatment diminished the incidence and severity of carditis. Treatment was more effective in preventing carditis in arthritis than in chorea (ascribed to the fact that salicylates are more universally used in arthritis than in chorea). Chorea treated by salicylates had a lower incidence of subsequent carditis than cases treated by arsenic, sedatives, or rest without drugs. Rest in bed for 8 to 12 weeks and salicylates in doses of 60 to 180 grains a day was the hospital routine for arthritis, all cases being treated alike regardless of severity.

Poynton (*Brit. Med. Jour.* 1925, 2:788) points out six methods of onset of rheumatism in the child. He believes the tonsils are an important site of infection, but does not think enucleation prevents a first attack of rheumatism, and rarely it may be followed shortly by a severe attack of carditis. Removal of unhealthy tonsils is definitely a valuable prophylactic step, but every case must be considered on its merits and not in a routine manner.

Raven (*Lancet*, 1925, 2:699) reports 92 cases of rheumatism, carditis, and chorea treated at St. Mary's Home, Broadstairs, with fresh air, sunlight, warmth, good food, and rest varying with the amount of activity. He feels that the results are very favorable when treated in institutions with the above methods.

515 cases of chorea have been analyzed by Burr (*Atlantic Med. Jour.* 1925, 28:568 and of these, 92 near relatives had suffered from it. Rheumatism is recorded as the most frequent disease occurring in the families—more frequent than chorea. Tonsillitis was the most frequent disease recorded. Burr does not believe that tonsillectomy is of value in preventing or curing chorea. He considers tonsillitis, growing pains, and acute articular rheumatism as very closely allied, if not different manifestations of the same poison. Vegetative endocarditis was the most constant necropsy finding.

Coates (*Brit. Med. Jour.* 1925, 1:550) and Coates and Thomas (*Lancet*, 1925, 2:326) point to the importance of finding the subcutaneous nodules in rheumatics with severe infection. They report 79.5% of cases as having nodules. This high percentage was found on careful examination, palpation of the spinous processes, and olecranon being particularly advised.

Hawkins (*N. Y. State Jour. Med.* 1925, 25:566) found that in a series of 215 cardiac

cases, 52.8% of the cases had infected tonsils, and 6% had purulent sinusitis. 62% of a group of rheumatic cardiac cases had infected tonsils, as compared with 29% in a control series of the same age.

Boas and Perla (*Amer. Jour. Med. Sci.* 1925, 170:529) report a study of 183 cases of mitral stenosis, showing that the condition is not uncommon in the 6th and 7th decades of life, 46 being over 50 years of age. It is in most cases the end result of a rheumatic infection in childhood or early life, in which the mitral narrowing is not extreme, and the lesion is non-progressive or slowly progressive. Women with such a condition may apparently undergo many pregnancies without distress or injury to the heart. In fourteen cases of mitral stenosis in elderly persons (over 60), the valvular lesion could not be interpreted as the end result of an old endocarditis, but must be considered as primary atherosclerosis of the mitral valve, and particularly of the mitral ring.

Loeb (*Med. Clin. N. A.* 1925, 9:455) summarizes the diagnostic points in acute rheumatic pericarditis. 34 cases are analyzed, one being reported at length.

Swift, Miller, and Boots (*Journal Clin. Invest.* 1924, 1:197) found leucocytosis to be concomitant with the severity of rheumatic fever, and they believe that counts made at frequent intervals give an approximate idea of the severity and duration of the infection.

An interesting report has been made by Telia (*Arch d. Mal du Coeur.* 1925, 18:531) of three cases of angina pectoris in young persons with mitral stenosis. One case is given in detail with autopsy findings, and two are yet alive. Telia concludes that the pain is caused by temporary compression of the left coronary artery between the dilated and hypertrophied left auricle and the dilated pulmonary artery.

Lutembacher (*Arch d. Mal. du Coeur.* 1925, 18:193) reports a case of aortic insufficiency with stenosis, of rheumatic origin, complicated by a prolonged form of ulcerous endocarditis, entailing the perforation of a semilunar valve and of the pars membranacea of the interventricular septum. Cabot (Case Records of the Mass. Gen. Hospital, BOSTON MED. AND SURG. JOUR. 1925, 192:699) reports a case of rheumatic aortic stenosis and regurgitation and mitral regurgitation in a man of 77 years, who had rheumatic fever at 14; heart disease of 63 years duration. Kessler (*Jour. Amer. Med. Assoc.* 1925, 86:32) gives an unusual case of enormous cardiac distension. The heart measured 12:5 inches across, and 7.5 inches perpendicularly. Mitral, aortic, and tricuspid lesions of rheumatic origin were present. The patient had auricular fibrillation, and failure many times, before succumbing 18 years after the heart was practically the above size.

Petty and Schaffer (*Jour. Amer. Med. Assoc.* 1925, 85:973) report a case of chorea insaniens in a girl of 12 years with recovery. The authors speak of Osler's two cases and the 12 in the literature.

"Diphtheria Heart"—Marvin (*Amer. Jour. Dis. Child.* 1925, 29:433) made an electrocardiographic and clinical study of 90 patients with diphtheria. The only abnormalities in the electrocardiograms which seemed to be of significance were disturbances in auriculo-ventricular or intraventricular conduction. Auricular and ventricular premature beats, sino-auricular block, and variations in the form of the P wave were seldom noted and appeared to have no significance. No evidence was obtained to indicate that in some patients the heart was primarily injured and in others the peripheral circulation suffered the first and greatest damage. Where there was circulatory impairment, the picture was that of myocardial failure. The signs of greatest value were vomiting, hepatic engorgement and a change in the character of the first heart sound. Necropsy in 5 of the 13 fatal cases revealed widespread myocarditis. Electrocardiograms were of value in diagnosis and prognosis.

Edmunds and Cooper (*Assoc. Amer. Phys.*, May, 1925, abstract *Jour. Amer. Med. Assoc.* 1925, 84:1596) studied the correlation between the Pharmacology and Pathology of Diphtheria Heart. Dogs were injected with diphtheria toxin in doses of 0.1 cc. at intervals of 2 to 3 days, the dogs surviving only 0.3 cc. at the most. Immediately after the pharmacologic experimental results were obtained, the hearts were examined pathologically. They showed the same features as were obtained in diphtheria in human beings. The hearts of these animals responded to the action of drugs as normal hearts do. Injection of digitalis was followed by an increase of blood pressure; epinephrin by a rapid and satisfactory increase in blood pressure; pituitary preparations also elicited a favorable response; salt solution was not very satisfactory; and 10% glucose was the most satisfactory. In dogs in which they could get 10% glucose into the circulation before the heart stopped, they had no fatality.

### "BACTERIAL ENDOCARDITIS"

"Acute Bacterial Endocarditis"—a study of 199 cases is reported by Thayer (*Weekly Roster and Medical Digest*, Mar. 14, 1925) with 138 autopsies. The following conclusions are drawn: Three-fourths of the fatal cases of rheumatic fever occur under 20 years. Early heart failure, rare in the bacterial cases, is common here. Rheumatic heart is a pancarditis, acute or adherent pericarditis in more than 95% of the fatal cases. Acute and subacute bacterial endocarditis are caused as follows: Staphylococcus albus 1%; influenza bacillus 4%; gonococcus 11¼%; staphylococcus aureus 13%; pneumococci

14%; streptococci 57%. Gonococcus endocarditis is nearly always fatal, running an acute course, but with longer duration than pneumococcus endocarditis.

"Subacute Bacterial Endocarditis"—Libman (*Weekly Roster and Med. Digest,* March 14, 1925) points to the four types of subacute endocarditis: Rheumatic, syphilitic, bacterial, and the indeterminate, formerly called cachectic. The subacute are more common than the acute, and 90% of the bacterial cases are due to non-hemolytic streptococci. He discusses the basis of making such a diagnosis.

Wright, H. D., (*Jour. Path. and Bact.* 1925, 28:541) from a study of the bacteriology of subacute infective endocarditis draws the following conclusions. When the blood culture is positive, bacteria are readily demonstrable in the valve lesion. In the nonbacteremic cases bacteria are present in the vegetations in the characteristic position, but they seem to differ somewhat in appearance from those seen in bacteremic cases, the differences being such as to suggest that they are undergoing disintegration. It is to be supposed then that the organisms may remain in sites for a long period without appearing in the blood stream, due to the fact that they are dead or prevented from reaching the blood stream by the physical barrier of the thrombus material separating them from it. Complete removal and subsequent healing by fibrosis still remains doubtful. Disappearance of the septicemia is by no means a cure of the disease which is always fatal in both septicemic and nonsepticemic cases.

From a review of 195 cases Wauchope (*Quart. Jour. Med.* 1925, 19:35) supports the view that this condition has become much more common since the recent war, believing that service in the army in some way predisposed to the acquirement of the disease. The point is not proven definitely, though the condition is undoubtedly found in many ex-soldiers who fall in the right age groups.

Landau and Held (*Bull. et. mém. Soc. Méd. d. Hôp.* 1925, 49:1322) call attention to the frequency of the positive Wassermann test in these cases, which they consider an incidental and secondary phenomenon. Of their 30 cases, the Wassermann test was positive in 10, including 6 absolutely free from syphilis, as confirmed by clinical examination and necropsy. Hess (*Münch. Med. Wchnschr.* 1925, 72:205) reports 27 cases. The Wassermann reaction was positive in 15 cases. Endothelial cells were found almost constantly in the first drop of blood dripping from the ear. The leucocyte count varied not only in different stages of the disease, but also according to the place (ear, finger, vein) where the blood was taken. Ceresoli (*Clin. Med. Ital.* 1925, 56:256) adds 9 new cases of endocarditis leuta to the 60 cases previously published in Italy.

"Gonococcus Endocarditis"—Three cases of gonococcus endocarditis are recorded, one each by Moreira (*Lisbon Med.* 1925, 1:545), Riecker (*Amer. Heart Jour.* 1925, 1:191), and Villela and Torres (*Sciencia Med., Rio de Janeiro* 1924, 2:703).

"Thyroid Heart Disease"—Foster (*Amer. Jour. Med. Sci.* 1925, 169:662) writes that the so-called cardiac deaths which follow operation, in cases of thyrotoxicosis, are seldom due to heart failure but to vascular failure; that is to say there is, in these cases, a vasomotor paralysis and a widening of the peripheral vascular field notable by a sharp drop in diastolic blood pressure, a condition similar to shock. Pardee (*Surg. Clin. N. A.* 1925, 5:11) reviews the field in general, especially emphasizing the value of quinidine in restoring and maintaining normal rhythm in thyroid hearts.

The status of the heart in myxedema has been the subject of several interesting reports during the past year. Fahr (*Jour. Amer. Med. Assoc.* 1925, 84:345) states that in myxedema there are definite objective signs as well as subjective symptoms of heart failure, which may be present for many years, do not respond completely to rest and digitalis, but are cured by thyroid medication. Characteristics of myxedema heart are an enormous dilatation of all the chambers of the heart, as proved by X-ray, and absence or negative activity of the T wave of the electrocardiogram in lead I. Under thyroid medication, the dilatations disappear and the T wave becomes positive. A few cases showed the split and prolonged Q-R-S group of delayed intraventricular conduction, and all showed a negative Q-R-S group in lead III. These changes also are corrected by thyroid. He reports three well marked and one mild case. Willius and Haines (*Amer. Heart Jour.* 1925, 1:67) fail to agree with Fahr from a study of 162 cases of high grade myxedema. In no case could they attribute heart failure or organic cardiovascular disease to myxedema, though they did find numerous electrocardiographic abnormalities which disappeared under thyroid medication. Christian (*Rhode Island Med. Jour.* 1925, 8:109) has seen no case that fully agrees with Fahr's description. He, however, states that there are signs and symptoms of circulatory insufficiency frequently. The therapy must be carefully planned since by elevating the basal metabolic rate, increased work is placed on the heart. Anemia may further increase the burden. It may be necessary hence to give digitalis and other cardiac therapy prior to thyroid treatment, or disaster from circulatory failure may occur.

"Syphilitic Heart Disease"—Warthin (*Amer. Heart Jour.* 1925, 1:1) gives a report based on the pathologic findings in 7 cases of sudden death. All cases showed a gross picture of myocarditis. Microscopically, there were local-

ized areas of infiltration throughout the myocardium, with either lymphocytes or many polymorphonuclear cells. In every one there was a subendothelial infiltration of cells of the large mononuclear type. In these areas, spirochetes were found in great numbers in every case. Sections of the aorta disclosed the presence of microscopic lesions, and spirochetes were found in all 7 aortas. Warthin calls attention to the fact that syphilis of the heart is the most common cause of death from syphilis, though it is rarely recognized either clinically or pathologically. Death usually occurs most commonly in the old fibroid heart in which there has been an atrophy of heart muscle and a gradual loss of compensation, and hence ultimately cardiac insufficiency arises as the result of the contractile condition on the fibers of the heart. Coronary syphilis may occur but is rare. This acute exacerbation of a chronic syphilis he considers an important form of syphilis.

Beretervide, E. A. and J. J. (*Arch. Lat. Amer. de Pediat.* 1925, 19:797) have studied the cadavers of 476 cases, and state that in children in whom the diameter of the ascending aorta was over 1 cm., up to the age of 8, and over 1.3 cm. from 9 to 14, congenital syphilis was present beyond doubt.

"Aneurysm"—There have been many interesting case reports, usually of rupture, and the number of reports of aneurysms of the splenic and renal arteries has been surprising.

Brockbank (*Brit. Med. Jour.* 1925, 2:606) reports a series of 56 cases of aneurysm, 47 men and 9 women. In about 95% there was a positive Wassermann test or a definite history of previous syphilis. The ages varied from 29 to 68 in the men, and 37 to 70 in the women. Of 200 cases of aortic aneurysm found post mortem, the average age at death was 38.7 years. Death occurred in these cases from exhaustion, heart failure, and rupture of the aneurysm with virtually equal frequency. DeGroodt (*Cercle Méd. Antwerp, Letter Jour. Amer. Med. Assoc.* 1925, 85:282) presented a comprehensive study on the multiplicity, development, and the mode of rupture of aneurysm of the aorta. Rupture occurs usually at the proximal end. A hardened thrombus capable of perforating the wall is usually found. DeGroodt condemns methods of wiring, and advised avoiding anything that favors the production of thrombi. A specimen of an aneurysm of a sinus of Valsalva giving rise to rupture of the left auricle was demonstrated.

Resnik and Keefer (*Jour. Amer. Med. Assoc.* 1925, 85:422) gave an interesting case report of dissecting aneurysm with signs of aortic insufficiency in which the aortic valves were normal. They suggest diminished coronary flow and increased work of the heart as the possible cause of the hypertrophy and dilatation found at necropsy. The aortic diastolic murmur they think possibly to have been due to weakness of the musculature supporting the aortic ring. Duftschmid (*Wien. Klin. Wchnschr.* 1925, 38:119) reports a case of dissecting aneurysm reaching from the valves of the aorta to its division, in which the patient died with symptoms of apoplexy.

Shennan (*Edinburgh Med. Jour.* 1925, 32:325) cites two cases, one of perforation of an aneurysm of the aorta into the superior vena cava, and one of perforation of an extension of an aneurysm of the aorta into the upper part of the right auricle. Shennan and Niven (*Jour. Path. and Bact.* 1925, 28:390) report a case of large cardiac aneurysm, occurring in the left ventricle, and being caused by endarteritis of the anterior descending branch of the left coronary artery.

Duguid (*Jour. Path. and Bact.* 1925, 28:389) reports a case of ruptured aneurysm of the basilar artery; Gluck (*Brit. Med. Jour.* 1925, 1:553) the intrapericardial rupture of an aortic aneurysm in an infant, aged 20 months, congenital syphilis was questionable; Dafoe (*Edinburgh Med. Jour.* 1925, 32:281) the rupture, in 2 cases, of an aortic aneurysm due to tuberculosis, and Jaffe (*Brit. Med. Jour.* 1925, 1:1173) the rupture of an abdominal aneurysm closely simulating acute intestinal obstruction.

McNealy and Spivak (*Surg. Gynec. and Obst.* 1925, 41:817) report 10 aneurysms in Scarpas's triangle, 7 being traumatic. In one of the traumatic cases syphilis may have been a factor and in the 3 spontaneous aneurysms the Wassermann was positive in each case. Davis (*Jour. Amer. Med. Assoc.* 1925, 84:200) reports a case of splenic artery aneurysm removed at operation. The aneurysm was saccular and 10 x 8 x 9 cm. with mural thrombosis. The Wassermann was negative, and there was no syphilitic history. Horsley (*Jour. Amer. Med. Assoc.* 1925, 85:189) reports a large aneurysm of the right axillary artery in a patient with bacterial endocarditis, who died with cerebral embolus.

"Arteriosclerosis"—From a study of 300 cases with arteriosclerosis, Mortensen (*Jour. Amer. Med. Assoc.* 1925, 85:1696) joins the ranks of those believing that arteriosclerosis or cardiovascular renal disease is definitely hereditary. He also ran a control group of 200 healthy women; studied 200 patients whose blood uric acid was below 3 mg. per 100 cc. and 200 with blood uric acid above 4 mg. per 100 cc. Mortensen believes that by heredity there is also an abnormal protein metabolism transmitted, manifest by an increased blood uric acid content, occurring long before any evidence of hypertension or renal disease is found. A reasonably low protein, basic diet, he considers as the most rational prophylactic measure.

Dow (*Brit. Med. Jour.* 1925, 2:162) dissected 8 bodies investigating to ascertain which vessels were most frequently and most extensively

affected with arterial degeneration. The abdominal aorta, common iliac, femoral and popliteal arteries were most frequently and more extensively affected than any other vessels. The mesenteric arteries were seldom involved. The internal carotid artery which lay in the cavernous sinus, and the part involving its bifurcation into the anterior and middle cerebral arteries frequently showed changes of a marked degree. The mitral valve and thoracic aorta were frequently involved; the tricuspid valve and pulmonary artery were not implicated in any of the specimens.

Mjassnikow (*Ztschr. f. Klin. Med.* 1925, 102:65 found a definite increase of the blood cholesteral regularly only in cases with arteriosclerosis in the aorta and the coronary arteries of an advanced stage. In those with milder forms of arteriosclerosis, it was increased only in 50% of the cases. 70 patients were studied. Kutschera-Archbergen (*Klin. Wchnschr.* 1925, 4:645) found a considerable increase in atheromatous vessels of lipoids which are insoluble in acetone.

Myslivecek (*Cas. lek. cesk* 1925, 64:732) reports a case of arteriosclerotic chorea in an elderly woman dying 12 days after the onset of the choreiform movements. Only chronic arteriosclerosis of the brain was found. Arrillaga (*Revista Méd. Lat.-Amer.* 1925, 10:1097) gives the clinical and microscopic aspect of Ayerza's disease, sclerosis of the pulmonary artery. He regards it as a primary affection, of syphilitic origin, while Ayerza assumes that it is secondary to some chronic pulmonary lesion.

"Angina Pectoris"—Sir James Mackenzie in the fourth edition of his book *Diseases of the Heart* (Oxford University Press, 1925,) deals at some length with angina pectoris making a distinction between primary and secondary angina, particularly as regards prognosis. He protests against cervical sympathectomy. Wearn (*Med. Clin. N. A.* 1925, 8:1593) gives a good summary of the present knowledge of angina pectoris, with emphasis on the symptoms and signs based on a study of 40 cases.

Penfield (*Amer. Jour. Med. Sci.* 1925, 170:864) gives a report of two cases treated by sympathectomy and gives an interesting discussion, with diagrams of the neurological mechanism of angina pectoris. He concludes that removal of a sympathetic ganglion removes the possibility of pain in the "motor" distribution of that ganglion only; pain is still possible in the motor distribution of the remaining ganglia; complete cervico-upper-thoracic sympathectomy abolishes the pain, but should be employed only where life is insupportable under best medical care; if operation is successful in abolishing pain, the patient should still be considered as having serious cardiac disease and treated accordingly. Danielopolu (*Comptes Rend de la Soc. d Biol.* 1925, 92:1155) also gives a good discussion with explanation of nerve roots and anastomoses involved.

Oberndorfer (*Münch. Med. Wchnschr.* 1925, 72:1495) discusses the anatomic findings in angina. In all typical cases of angina pectoris, he found anatomic manifestations of circulatory disturbances in that part of the left ventricle, which receives its blood supply from the descending branch of the left coronary artery, that is, the apex, septum, and sometimes the anterior wall. Anemic infarcts, scar formations and secondary aneurysms were found in these locations. He believes that temporary spasms of the artery may lead to anemic infarcts.

Gallavardin (*French Congress Med., Letter Jour. Amer. Med. Assoc.* 1925, 85:837) discussed the present hypotheses of angina, differentiating between certain types, particularly with reference to prognosis.

Leriche (*Presse Méd.* 1925, 33:1361) performed experiments on a man under local anesthesia, induced for sympathectomy. Electrolization of the left stellate ganglion caused intense pain and oppression in the precordial region. The phenomenon subsided after the injection of procain in the same ganglion. In 2 other cases the current applied to the upper part of the left first thoracic ganglion caused sharp pain in the corresponding arm. Applied to the lower part of the ganglion, caused pain in the heart. In another case, an attack of angina occurred during sympathectomy for angina pectoris, and the paroxysm was relieved by injection of procain in the left inferior cervical ganglion.

Askanazy (*Virchow's Arch. Path. Anat.* 1925, 254:329) reports the sudden death of a man, 33 years of age, who was known clinically to have had a large heart and auricular fibrillation. Inflammation and perineural infiltration with cells of the cardiac nerves, not the conduction system, was found at autopsy. Askanazy suggests this as a possible cause of some cases of heart pain or sudden death.

"Coronary Disease"—Christian (*Amer. Heart Jour.* 1925, 1:129) gives the clinical picture of the rather common clinical condition of cardiac infarction, which he says is now easily recognized by fourth year medical students, as compared with its rarely being recognized clinically a few years ago. Willius (*Med. Clin. N. A.* 1925, 8:1181) gives a clinical lecture using one case carefully followed by electrocardiograms. The tracings showed right bundle branch block, alternating nodal premature contractions, large exaggerated T waves in leads II and III, then negative T waves varying from day to day. Levine (*Med. Clin. N. A.* 1925, 8:1719) reviews the clinical syndrome of angina pectoris and coronary occlusion, speaking of the latter as cardiac infarction. 5 typical occlusion cases were reported that recovered

and were restored to previous activity. Pletnew (*Ztschr. f. Klin. Med.* 1925, 102:295) and Nathanson (*Amer. Jour. Med. Sci.* 1925, 170:240) review the field thoroughly. The latter attempted to construct a clinical picture of coronary sclerosis by an analysis of a series of fatal cases coming to necropsy.

Werley (*Texas State Jour. Med.* 1925, 21:428) reports that of 200 necropsies, 25% had badly diseased hearts and aortas. 4% showed cardiac infarct or cardiac aneurysm. Three cases showed rupture of the left ventricle, following coronary obstruction. In 3 cases the right coronary artery was the occluded one. Gager (*Jour. Amer. Med. Assoc.* 1925, 84:1730) calls attention to the fact that a sudden fall in arterial tension, following severe cardiac pain, rests on the physiologic basis of infarction and myocardial insufficiency following an occlusion.

Kretz (*Wien. Arch. f. inn. med.* 1925, 9:419) found little parallelism between the clinical history and the anatomic findings in 106 cadavers. He agrees that frequently clinical symptoms are mistakenly attributed to sclerosis of the coronary arteries. 6 out of 16 cases of extreme sclerosis and narrowing of the lumen had had no cardiac symptoms during life.

Willius and Barnes (*Jour. Lab. and Clin. Med.* 1925, 19:427) report 9 new cases and review 24 published cases as regards electrocardiographic study, and Willius (*Atlantic Med. Jour.* 1925, 29:9) reported 2 further cases in a separate communication. Abnormalities of the T wave occurred in 8 cases. Negativity of the T wave in one or more leads was the most frequent change, and at times the changes varied while under observation. In two cases the R and T wave were almost fused, occurring as a transient manifestation. Kerr, Larkey, and Larsen (*Cal. and West. Med.* 1925, 23:46) report 15 cases all in men, 11 being over 40 years of age. Two cases showed subacute bacterial endocarditis—infarction by embolism.

Fulton (*Amer. Heart Jour.* 1925, 1:138) discusses the mechanism of death in coronary thrombosis. He divides the deaths into 3 groups: (1) the deaths in which the immediate cause is more or less remote from the original lesion are not strictly cardiac deaths, usually being the result of embolism from intracardiac thrombosis; (2) damage to the heart as the result of the cutting off of the blood supply may lead to progressive weakening of the heart muscle, and death occurs as it does in other instances of myocardial failure; (3) the heart ceases to beat because of some interference with the mechanism with which the heart beat is maintained in its orderly sequence. The most important causes of sudden death are heart rupture, ventricular fibrillation, pulmonary embolism, and heart block.

Jamison, and Hauser (*Jour. Amer. Med. Assoc.* 1925, 85:1398) report an extremely interesting death from coronary occlusion at the age of 18 years. At necropsy the heart weighed 240 gm. and the coronaries were tortuous and rigid. Martin and Waugh (*Ann. Clin. Med.* 1925, 4:183) report the rupture of ventricular septum secondary to thrombosis of the left coronary artery.

"Pericardium"—Murphy (*Jour. Amer. Med. Assoc.* 1925, 84:1950) considers adhesive mediastino-pericarditis as a common sequel of the recent pan-epidemic of influenza and pneumonia. Rigler (*Jour. Amer. Med. Assoc.* 1925, 84:504) collected 72 cases of pneumo-pericardium, most of them complicated by the presence of fluid. The causes are: (1) spontaneous gas production; (2) traumatic perforation of the pericardium; (3) perforation from or into a neighboring organ; (4) artificial injection of gas. Knox (*Amer. Jour. Med. Sci.* 1925, 169:897) reports a case of chronic mediastinitis, which caused the death of the patient, in which syphilis is believed to have been the etiological factor. Rawls (*Amer. Jour. Med. Sci.* 1925, 169:815) cites a case of tuberculous pericarditis, in which the process proved to be primary in the pericardium.

"Hypertensive Heart Disease"—Fishberg (*Arch. Int. Med.* 1925, 35:650) discusses the anatomic findings in essential hypertension from the necropsy findings in 72 cases. All showed lesions of the terminal arterioles, the minute arterioles of the kidneys being always affected. The distribution was quite different from large vessel atherosclerosis. Generalized arteriosclerosis does not exist in essential hypertension. Skin, muscles, myocardium, lungs, thyroid, and gastro-intestinal tract are rarely involved. Evans (*Brit. Med. Jour.* 1925, 2:1167) states that hyperpiesia has a morbid anatomy by which it can be recognized after death. These changes are: (1) left ventricular hypertrophy; and (2) small arteries and arterioles show "diffuse hyperplastic sclerosis".

Miscellaneous—Cardiac Muscle in Hypertrophy and Atrophy. The state of cardiac muscle in hypertrophy and atrophy is reported by Karsner, Saphir, and Todd (*Amer. Jour. Path.* 1925, 1:1351). Three hearts were carefully studied. The normal heart weighed 300 gm., the hypertrophic 500 gm., and the atrophic 165 gm. More than 2,000 accurately measured fields were studied in each heart. The authors conclude that in hypertrophy the increased bulk is due solely to an increase in the size of the preexistent muscle fibers. In atrophy there is a decrease in the size of the fibers and an actual disappearance of the fibers, due, in all probability, to the autolytic processes, studied by Bradley and his collaborators. There is also in atrophy an increase in the number of nuclei. In both conditions there is an approach toward uniformity in the size of the fibers, which rep-

resents an entirely new conception of variability and adaption.

"Auricles in Auricular Fibrillation"—Frothingham (*Arch. Int. Med.* 1925, 36:437) studied at post mortem examination 11 cases with auricular fibrillation, and 23 cases with normal rhythm with especial reference to auricular pathology. He found no typical lesion in the auricles that could be looked on as the cause of auricular fibrillation, although a slight degenerative lesion was found in the muscle fibers of the auricles much more frequently with auricular fibrillation than in regular beating hearts of the same age. There is no way of telling whether this lesion was the result of the the irregularity or a factor in its production.

"Mental Symptoms in Cardiovascular Disease"—Among several hundred mental patients, Stone (*Ohio State Med. Jour.* 1925, 21:92) found 40 certain cardiovascular disturbances which seemed to bear a direct relationship to the disordered mental conditions for which they were primarily admitted. The mental symptoms dominated the clinical picture, yet the cardiovascular system could be definitely connected as an explanation for the presence of the symptoms. Renal disease, syphilis, and alcoholism were excluded from consideration.

"Heart in Prostatic Hypertrophy"—Willius (*Jour. Urology* 1925, 13:337) gives a report of the study of 705 patients with prostatic hypertrophy special investigation being made of the cardiovascular system, including electrocardiograms. 293 had cardiovascular disease. Arteriosclerotic type of cardiac disease occurred in 125 cases, 106 also having hypertension. 50 cases showed hypertension without arteriosclerosis. 22 patients had angina pectoris, in one case associated with syphilitic aortitis. There were a few cases of chronic rheumatic endocarditis, and one of myocardial degeneration associated with exophthalmic goiter.

"Liver Disease caused by Cardiac Defects"—Brooks (*Med. Clin. N. A.* 1925, 9:311) discusses 3 cases with large livers as well as other difficulties, in which the heart was not at first considered the cause, later proving to account for them by engorgement. He calls attention to the frequency with which the liver is involved in cardiac conditions, at times being overlooked. and at others disguising the cardiac picture.

"Pregnancy"—Bierring (*Ugesk. f. Laeger. Copenhagen* 1925, 87:95) has had 87 obstetric patients with complicating heart disease, a total of 107 childbirths. Only 11 of the women developed severe heart symptoms, fatal in 6 cases, all elderly multiparas. Fully 50% passed through the pregnancy without symptoms. 5 of them had recurrences of acute articular rheumatism, fatal in one. Failing compensation predisposes to spontaneous interruption as shown by 11 cases of abortion. Of the 107 pregnancies, there were 98 viable children. Frey (*Klin. Wchnschr.* 1925, 4:577) regards the increase in the volume of the blood in pregnancy as the most important cause of strain on the heart.

"Pertussis"—Ledbetter and White (*Jour. Amer. Med. Assoc.* 1925, 4:1022) review the literature on the subject of the effect of pertussis on the heart;—232 cases reported by the authors. The experience of pediatricians and the literature do not yield proof of endocardial or myocardial damage during or after pertussis. They agree that there is undoubtedly a temporary mechanical strain on the right side of the heart, though circulatory failure does not seem to occur. In their series, they found no evidence that the heart was affected as a result of pertussis.

"Tumors"—Cabred and Mosto (*Prensa Méd. Argentina* 1925, 11:1194) comment on the rarity of the heart as a site for tumor. They review the literature, and state that rarely is a diagnosis made before necropsy. 11 cases are reported by the authors. Fibromas form the majority of the tumors. Achard and Mouzon *Bull. et mem. Soc. Méd. d Hop. de Paris* 1925, 49:108) report a case of secondary cancer in the heart in a woman, age 64 years, with right ventricular intramyocardiac nodules. Beck and Thatcher (*Arch. Int. Med.* 1925, 36:830) report a case of primary sarcoma in the wall of the left auricle with metastases to the intestines. They state that there are 150 cases in literature of primary cardiac neoplasms.

"Rupture of the Heart"—Locke (*Med. Clin. N. A.* 1925, 8:1669) goes into full details of the subject, adding 2 new cases. He found 71 cases in the literature, and there was coronary thrombosis in 25 of the 71. Lewis (*Ann. Clin. Med.* 1925, 4:36) reports 7 cases with full necropsy reports. Krumbbaar and Crowell (*Amer. Jour. Med. Sci.* 1925, 170:828) give a clinicopathologic study based on 22 unpublished cases of spontaneous rupture of the heart. 18 of the original cases had coronary sclerosis, and some thrombosis. No note was made as to the condition of the coronaries in the remaining 4 cases. Skeen and Dow (*Brit. Med. Jour.* 1925, 1:262) report a case of rupture due to an almost complete obstruction of the right coronary artery.

Howard (*Jour. Amer. Med Assoc.* 1925, 84:1952) reports an interesting case of aortic insufficiency due to rupture by strain of a normal aortic valve, the patient dying 11 days later with severe failure. Maitland (*Brit. Med. Jour.* 1925, 1:69) reports a case of spontaneous rupture of the thoracic aorta.

"Emphysema"—Jagic and Spengler in their short book "Emphysem und Emphysemberz" (*Vienna: Julius Springer,* 1924) describe with some emphasis the heart condition attending emphysema.

# Case Records of the Massachusetts General Hospital

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICO-PATHOLOGICAL EXERCISES

EDITED BY
RICHARD C. CABOT, M.D., AND HUGH CABOT, M.D.
F. M. PAINTER, A.B., ASSISTANT EDITOR

## CASE 12171

### SYMPATHECTOMY FOR ANGINA PECTORIS IN A WOMAN OF TWENTY-TWO

### MEDICAL AND SURGICAL DEPARTMENTS

A married Italian woman of twenty entered May 2 1923 complaining of pain in the left arm and left chest, shortness of breath and weakness.

When she was six years old she was ill four months with "rheumatic fever." Practically all the joints were involved. During the next three or four winters she had "rheumatic" attacks. From the age of ten until two years before admission she was well. Then she began to have pain in the left arm, dyspnea on mild exertion, palpitation and slight unproductive cough, and was "feverish" and weak. She was in bed two months. In September, four months later, she was ill for a week with fever, malaise and slight cough. In March 1922 she had sore throat and symptoms of a "cold", and again had pain in the left arm and left chest, front and back, dyspnea, palpitation and cough. The left knee also was swollen and painful, and the feet slightly swollen. She was in bed two months. Four months before admission she again had sore throat for several days, and after working two weeks had to stop on account of recurrence of the pain, which came in attacks especially at night and lasted sometimes many hours. After the attacks the left arm felt weak and tired. She also had palpitation, cough, sometimes with yellowish sputum, occasional feverish feeling, and some sweating at night, especially of the head. Her appetite was diminished. Her bowels required laxatives. She had lost ten pounds in the past two years. She felt weak.

Her family history was unimportant. Her past history was negative.

Examination showed a well nourished young woman with highly flushed cheeks, fairly comfortable flat in bed. The whole body was shaken by the heart pulsations. In the right supraclavicular fossa was a movable gland, not tender, the size of a walnut. The apex impulse of the heart was easily seen and felt in the 6th space 13 cm. from the midline. The percussion measurements are shown in Figure 1. A heaving forceful pulsation was felt all over the chest and upper abdomen, visible in the neck. The aortic second sound was accentuated. A thrill was palpable at the aortic area and less definitely at the apex. There was a loud harsh systolic murmur at the aortic area and the apex, heard also all over the precordia, and a softer blowing diastolic heard at all areas. An electrocardiogram showed normal rhythm, rate 100, inverted T wave in lead II, marked left axis deviation. The pulse was Corrigan, felt easily along the arms and legs. The blood pressure was 165/0. The extremities showed slight tremor. There was no cyanosis, edema or ascites.

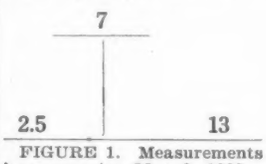


FIGURE 1. Measurements by percussion May 2, 1923.

*Laboratory data.* Urine cloudy at one of six examinations, specific gravity 1.014-1.020, amount normal, the very slightest possible trace to a trace of albumin at five examinations, rare red blood corpuscles at five; the sixth specimen, taken by catheter, showed 1-2 leucocytes per high power field, no red cells; on culture a slight growth of staphylococci. Two other specimens showed leucocytes. Blood: 10,200-13,200 leucocytes, 40 per cent. polynuclears, 75-85 per cent. hemoglobin, 4,400,000 red cells, smear normal. Wassermann negative. Non-protein nitrogen 27.6 mgm. per 100 c.c.

X-ray showed the frontal sinuses unusually small. Both antra were slightly less radiant than normal, but not definitely pathological. Teeth negative. The heart shadow was very much enlarged in all diameters (see Figure 2),

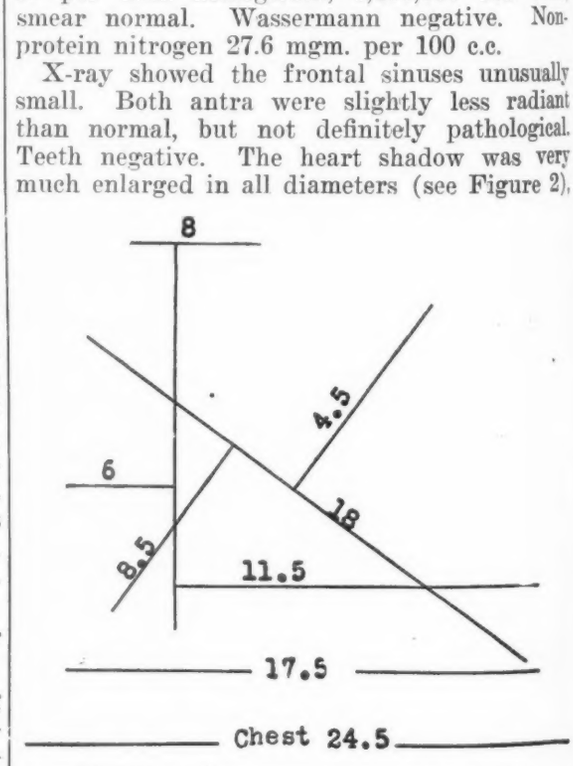


FIGURE 2. Measurements by X-ray May 8, 1923.

and lay rather horizontally in the chest. The supracardiac dullness was increased.

The temperature was 99.7° the day of admis-

sion; later 98°-99.2°. The pulse was 72-100, with one rise to 120 May 12. The respirations were normal.

The patient was digitalized without change in the pulse rate. Nitroglycerine relieved the pain in the arm somewhat. By May 7 there was no joint pain. There were two or three attacks of very rapid pulse associated with chest and arm pain.

A throat consultant reported septic tonsils, and advised tonsillectomy.

May 19 she was discharged to another hospital, where she remained until the following November, feeling fairly well. Her pain was not frequent. She gained about twenty pounds.

After she returned home attacks of knife-like pain over the lower precordia, variably transmitted to the left back, the axilla or the left arm occurred almost every night, waking her and forcing her to sit up to catch her breath. The attacks were short and left her feeling weak.

January 10 1924 she reëntered the hospital.

Examination now showed slight cyanosis of the lips. The heart was rapid, regular, with a diffuse heaving apex reaching to the anterior axillary line in the fifth space. The percussion measurements are shown in Figure 3. There was a systolic thrill at the apex and a double thrill at the aortic area. At the apex was a snapping first sound with blowing systolic and diastolic murmurs, heard over the precordia and fusing with two-way murmurs at the base. The aortic second sound was absent, replaced by a blowing diastolic murmur. The systolic murmur at the aortic area was loud, rough, and transmitted to the neck. The pulmonic second sound was not heard. Corrigan pulse. Pistol-shot at ankle. Visible pulsation in neck. Electrocardiogram showed during an attack of pain marked sinoauricular tachycardia, rate 140; after attack of pain rate 110. Normal rate 90. Markedly inverted $T_2$ wave. The rest of the examination was as before.

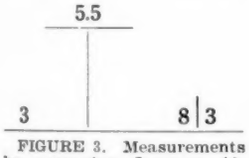



FIGURE 3. Measurements by percussion January 10, 1924.

*Laboratory data.* Urine alkaline at one of three examinations; 1-2 red cells in that specimen only. Leucocytosis now 13,000-18,600 in 9 specimens, 8,400-10,800 in 7. Polynuclears 72 per cent. Wassermann negative.

The temperature was 96.8° to 99.5°, the pulse 72 to 140, the respiration 18 to 30.

During Dr. White's examination the patient suddenly complained of precordial pain referred down her left arm to her finger tips. At the same time he found that the apex pulse rate had risen sharply from 90 to nearly 140, regular. She continued to have similar attacks of severe pain fairly often.

An electrocardiogram taken at 3:55 a. m. January 13, during an attack, showed auricular tachycardia, probably sinoauricular, rate 140. At 4:10 vagal pressure, right and left, and eyeball pressure did not alter the rate or stop the tachycardia, which ceased at about 4:20. She felt the pain in the axilla and along the right border and angle of the scapula disappear at the same time. Then the arm became less numb. Some tenderness persisted and palpitation continued for about ten minutes, gradually disappearing. At 4:45 she thought the heart much quieter, had no pain and only very slight tenderness in the areas mentioned. An electrocardiogram at 4:25 showed normal rhythm, rate 120. There was a gradual lengthening of the T-P interval (diastole) in the course of the recording of three strips of lead II. In a third strip the rate was 110. An electrocardiogram at 4:36 showed normal rhythm, rate 90-100, gradual lengthening of T-P interval.

January 18 she had nosebleed. January 23 quinidine therapy was started, gr. iii twice a day.

January 25 she had several attacks of tachycardia with severe precordial pain radiating down the arm. During the attacks there was definite hyperesthesia to pin prick and to cotton wool over the side of the arm. Nitroglycerine gave relief. The systolic blood pressure was 220, the diastolic 40 at the time of the pain. The paroxysms recurred during the next two days with fairly severe pain. There was marked heaving of the precordium and striking pulsation of the vessels of the neck. Capillary pulse was easily noticeable in the fingers. January 29 nitroglycerin was tried during an attack. The systolic blood pressure dropped from 200 to 140. The pulse slowed from 130 to 90. The pain disappeared. It was thought the attack was definitely cut short. The patient now volunteered that when nitroglycerin was taken at the beginning of an attack, when pain and tachycardia were first felt, the pain disappeared within a minute or two, though the tachycardia continued for the usual time, fifteen minutes to a half hour.

Beginning February 12 she had pain in the right thigh and knee, later in the back, the left thigh and knee, and the muscles and joints of the right shoulder, arm, leg and neck. Sodium salicylate therapy was started February 19. By the 22nd she had no more sharp pain in these regions except a little in the lumbar region.

February 22 she awoke with an attack of precordial pain at 11:35 p. m. At 11:42 the pulse was 122.

11:45. Electrocardiogram showed normal rhythm, rate 120.
11:51. Blood pressure 175/40-20.
11:52. Nitroglycerin 1/100 under tongue.
11:54. Pulse 132.
11:55-11:56. Blood pressure 170/30. Pain suddenly disappeared.

11:57. Pulse 104.
11:58. Pulse 102.
11:59. Blood pressure 120/20.
12:00. Blood pressure 135/30-20.
12:04. Blood pressure 175/30-20.
12:15. Electrocardiogram showed normal rhythm, rate 100.

The pain was at first substernal, then radiated to the precordium, the left shoulder, the angle of the left scapula and the left arm along its posterior aspect and only as far as the elbow.

She continued to have slight attacks of "anginoid" pain, one every night. March 1 she had sore throat. A culture showed streptococci. March 7 during a mild attack of "anginoid" pain a tenth of a grain of calomel was given for placebo at 10:38.

10:40-10:43. Blood pressure 210/60.
10:46. Blood pressure 208/60.
10:48. Blood pressure 200/50.
10:51. Pain diminishing.
10:53. Blood pressure 190/45. Pain suddenly disappeared.

March 20 calomel was given at 7:43, during a sharp attack of pain.

7:46. Blood pressure 215/60.
7:47. Pulse 112.
7:49. Pain as strong as ever.
7:50. Pain diminished while the blood pressure was being taken, 180/70.
7:51. Pulse rate 90.
7:52. Pain was gone.
8:58. Another attack began.
8:60. Blood pressure 210/60.
8:61. Nitroglycerin gr. 1/100 under tongue.
8:62. Blood pressure 170/50. Pulse 116.
8:63. Pain less severe.
8:64. Pain gone.
8:65. Pulse 104.

April 1 1924 examination showed good color, mitral facies, slight obesity. Apex impulse forceful, maximal in the fifth space 10.5 cm. to the left of midsternum and 2.5 cm. outside the midclavicular line. No increase in cardiac dullness to the right or in supraclavicular dullness. The first sound at the apex was almost entirely replaced by a rough systolic murmur. The second sound was accentuated and followed by a rough diastolic murmur filling diastole. At the apex a diastolic thrill was felt. At the base a marked diastolic thrill was felt all over the manubrium, maximum in intensity in the aortic area, where rough systolic and high pitched blowing diastolic murmurs were loudest. Both systolic and diastolic murmurs could be heard at any point in the chest, their quality depending upon the position of a given point with relation to the apex or aortic area. The blood pressure was 170/55. There was no tenderness of the muscles or joints and no edema of the ankles after the patient had been up all morning. That day she was discharged to the Cardiac Clinic.

Records of the Cardiac Clinic show that she continued to have mild attacks of angina and dyspnea on climbing one flight of stairs. April 17 the blood pressure was lower than it had ever been found before, 140/30. The left sternoclavicular junction and the clavicle were more prominent than the right. A prescription was given for aspirin gr. xxx daily until the rheumatic pains disappeared; otherwise to continue régime of several hours up and about. She did well on this régime, and by August was feeling better and had less frequent attacks. September 25, however, the pain was almost continuous and nitroglycerin gave only short relief. The aortic systolic murmur was very harsh.

Beginning in January 1925 she was in bed six weeks with polyarthritis and fever. For the first time she had some edema of the ankles, passing away at night. Pistol shot could be heard over every artery. In August she was better than she had been for months. She still had three or four attacks daily of sharp pain in the left arm radiating to the precordium. The right radial pulse was more typically Corrigan than the left, which had an obvious anacrotic wave. With a drop of pulse to the rate of 80 when she was resting in the recumbent position the

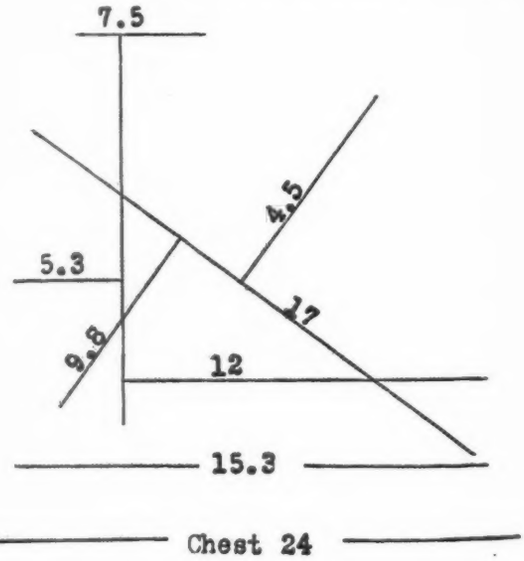



FIGURE 4. Measurements by X-ray October 15, 1925.

blood pressure changed as follows: right arm 165-155-140/50-30-70, left arm 120-130-130/30-20-90. The pulse pressure decreased, but the difference in the pulse and blood pressure persisted. The apex impulse was maximal in the sixth space. No edema.

October 15 the pain was almost constant. She had used two hundred nitroglycerin pills in a week and a half. X-ray showed the heart shadow about as it was at the last examination, if anything slightly smaller. (See Figure 4.)

January 28, 1926, it was found that during

the year the dyspnea had grown somewhat worse; she had had no edema, and only one nosebleed, two weeks before the visit. For two months she had used two pillows. She had had occasional attacks of dizziness, not associated with angina, and occasional headache.

January 28 1926 she was admitted to the wards for the third time.

Examination showed her sitting up in bed breathing slightly rapidly but in no discomfort. There was no cyanosis. In the right neck posterior to the sternocleidomastoid was a firm, non-tender movable mass approximately 4 by 3 by 2 cm. deep. The apex impulse of the heart was seen and felt in the sixth space. The percussion measurments are shown in Figure 5. At the apex were diastolic and systolic thrills. There was a loud rough systolic murmur lasting throughout systole and replacing the first sound, and a blowing diastolic transmitted to the axilla. At the first right interspace was an extremely harsh loud systolic murmur practically obliterating the second sound, and a soft diastolic transmitted to the neck. There was marked venous pulsation in the left supraclavicular fossa. There was an occasional extrasystole, Corrigan pulse, pistol shot in the femorals; blood pressure 175/30. Electrocardiograms showed normal rhythm, rate 100, marked left axis deviation, as before.

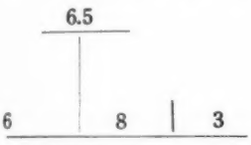



FIGURE 5. Measurements by percussion January 28, 1926.

*Laboratory data.* Urine showed no red blood cells and was not remarkable. Blood normal. Wassermann not recorded; it had been negative in 1923 and 1924.

Before operation the temperature was 98.2° to 100.4°, the pulse 80 to 120, the respiration 20 to 30.

On February 5 the left superior cervical ganglion was excised. The patient made a good operative recovery. She had considerable hyperesthesia over the left side of the neck extending to the scalp. She did well, but complained of pain in the back and around the costal margin over the liver. Examination was negative. February 11 half the stitches were out. There was less pain, but the hyperesthesia persisted. February 13 all the stitches were out. Hyperesthesia was the only complaint. February 16 she was feeling well and had no more pain. There was tenderness over the left side of the neck, head and face. February 17 she was discharged to the Cardiac Clinic.

In the middle of March she was sick at home with a bad cold, but was having no more angina pectoris.

## Discussion

BY PAUL D. WHITE, M.D., AND EDWARD P. RICHARDSON, M.D.

Dr. White: The first point of importance in this case is the *rheumatic* history, which is undoubted, and which is so common in the children coming to our hospital clinics in New England.

The second point of importance is the evidence of *valvular disease* on examination. The murmurs indicate disease of both aortic and mitral valves with cardiac enlargement.

Finally the third and most important point of all is the occurrence of *attacks of heart pain, anginal in character and associated with palpitation due to a regular tachycardia.* It was because of this symptom that this case was selected for *discussion both of diagnosis and of treatment.*

I well remember my first conclusion as to the cause of the attacks of pain and rapid heart action. Because of her age (twenty years) and apparent nervousness I thought at first that she was highly neurotic and had spells of nervousness with pain and tachycardia. Then when I saw her during an attack I realized that the pain and palpitation were marked, with very sudden onset and not to be ascribed simply to nervousness. For these reasons and again because of her age and also because of the rapid pulse (140) it seemed likely that paroxysmal tachycardia in short attacks associated with rheumatic heart disease would best explain the situation.

After more detailed study of the paroxysms of pain and tachycardia, however, some of which is listed in this report, it became apparent that the only possible explanation of the phenomenon was angina pectoris. The pain was always the very earliest symptom, it was very severe, much more so than I have ever seen with paroxysmal tachycardia, it was always relieved completely in two to three minutes by nitroglycerin and not by placebos, and it was distributed like that of angina pectoris to the left arm, even to the finger tips. It was associated as angina pectoris frequently is with temporary tachycardia and hypertension. But most important of all, electrocardiograms taken during paroxysms of pain did not show definite paroxysmal tachycardia, that is, there was no clear evidence of ectopic origin of the auricular impulse. Finally the heart rate was somewhat variable, not always the same and also not so high as it usually is in repeated paroxysms of paroxysmal tachycardia. During the pain the heart rate varied from 140 to 90, registering in different attacks 140, 132, 120, 116, 112, 108 and 90. The rise in blood pressure was also variable, in different attacks registering 215, 210, 190 and 175 millimeters systolic. On one occasion the systolic blood pressure dropped to 120 after relief

by nitroglycerin. The diastolic blood pressure was low because of the presence of aortic regurgitation.

Although angina pectoris is commonest in middle age and old age it may rarely occur in young people. It has been reported in other cases of rheumatic heart disease with aortic valve involvement, The mechanism still remains obscure, but must be some transient functional disturbance. On several occasions in this patient, excitement, as when a death occurred in the ward or when she was examined by a group of medical students or doctors, was evidently responsible for the anginal attacks. Many attacks occurred at night during sleep, perhaps from the excitement of dreams.

The patient was discharged from the ward April 1 1924 and thereafter for the next two years made occasional visits to the Cardiac Clinic of the Out-Patient Department, where she showed no appreciable change in physical examination but reported daily attacks of angina pectoris, at times almost continuous and much relieved by nitroglycerin. Rheumatic pains and especially the attack of acute rheumatic fever in January 1925 suggest that a persistent rheumatic infection in the heart may in some way have been a factor in the production of the heart pain. Salicylates however were ineffective in treatment of the pain. In October of last year she used about two-hundred tablets of nitroglycerin, 1/100 grain each, in ten days' time.

The miserable existence this young woman was leading, the careful study of the attacks of angina pectoris, the favorable condition for operation, and the failure of medical treatment to prevent the heart pain caused us after much consideration to recommend cervical sympathectomy by Dr. Edward P. Richardson in this case. Accordingly she was admitted in January 1926 to the wards for the third time for operation, which was done on February 5. The left superior cervical ganglion was removed without incident. There was no more anginal pain.

A report from home about six weeks after the operation (middle of March) stated that there had been no return of the angina pectoris. She was sick at the time of this report with a bad cold. It was of course too early to tell the final result of the cervical sympathectomy in this case, but to date the report was encouraging.

### PRE-OPERATIVE DIAGNOSIS

Angina pectoris.

### OPERATION

Local novocain infiltration of superficial cervical plexus behind the sternomastoid muscle and of line of incision near anterior margin of same muscle. Sternomastoid retracted outward and carotid sheath inward. A line of somewhat enlarged lymph nodes with inflammatory changes in areolar tissue was met. Clear dissection was difficult. Cervical sympathetic nerve finally found adherent to posterior aspect of jugular vein, separated and followed upward and finally removed. No effect on pulse was noted. Patient complained of moderate pain during excision of ganglion, the pain apparently not so severe as that caused by suture of the skin. Wound closed without drainage. Whole enlargement of the left superior ganglion was removed.

### FURTHER DISCUSSION

DR. RICHARDSON: The demonstration by Dr. White of true anginal pain in this patient, the frequency and severity of the attacks, brought into consideration an attempt to relieve the pain by surgery. While the actual course of the afferent impulse causing pain is uncertain, it seems probable that it is transmitted through the sympathetic nerve fibres and ganglia, ordinarily chiefly through those of the left side. The pioneer in operating on the sympathetic system for angina pectoris was Jonnesco, who recommends removal of the whole cervicothoracic chain, first on the left, but if necessary on both sides. In this country Coffey and Brown, and more recently Kerr, have reported in the majority of cases good results from removing the left superior ganglion only.

The questions to decide in this case are, what is the risk of operation, what are the possibilities of harm, and what is the likelihood of benefit? With careful selection of cases the mortality of operation in angina pectoris is not prohibitive according to the literature, although I have had an unfortunate experience in this respect. In nine operations on six patients there have been two deaths, one from ether anaesthesia, and one a sudden death eight hours after operation in a patient apparently in good condition. Syphilitic cases are apparently poorer risks. In this young woman the risk of operation should be slight.

Operation in this case was slightly more difficult than usual on account of the old adenitis. Since the sudden death above described we like these patients to have a special nurse during the first forty-eight hours after operation, so that if distress occurs amyl nitrite may immediately be given.

The eye changes caused by operation, narrowing of the fissure and contracture of the pupil. are not a serious disability. In occasional instances removal of the superior ganglion produces a neuralgic pain in the side of the face which is persistent and distressing.

Our results have not been so good as those usually described in the literature. Of five patients surviving the removal of the superior ganglion three have received no relief, one has

been considerably improved, and the fifth is the present case. All these cases were apparently benefited during their stay in the hospital, but the pain returned as before on discharge. One patient has neuralgic pain in the jaw.

If this patient has a return of her attacks in the old severity, removal of the whole chain on the left side including the inferior cervical and first thoracic ganglion may be considered, in order to cause a more extensive blocking of the afferent impulses.

We have no reason to be enthusiastic about the results of operation from our experience in this clinic, but feel from reports in the literature and our own instances in which operation has been effective that we are justified in continuing with a limited operation in selected cases.

LATER NOTE

April 1 1926 the patient's sister was seen at the Out-Patient Department and reported that following an attack of influenza the patient had been having a return of angina, relieved by nitroglycerin.

April 15 1926. Patient seen again in the clinic in the Out-Patient Department. About one month ago she had an acute respiratory infection during the convalescence from which she began again to have typical angina pectoris relieved by nitroglycerin. At present she requires two or three tablets daily (having used about fifty in the past two weeks), which is one-half or less than half the amount that was necessary before the operation. Also two weeks ago at home she had edema of legs and abdomen, now almost gone after rest alone. This apparently was the result of congestive failure.

Physical examination showed the same cardiac findings as previously—that is, aortic stenosis and regurgitation, mitral stenosis and regurgitation, cardiac enlargement, normal rhythm and slight congestive failure (lungs clear, but slight edema over the shins). The left pupil was contracted. The blood pressure was 195 mm. mercury systolic and 30 mm. diastolic. The pulse rate was 106. Temperature 99°.

DIAGNOSIS

Rheumatic heart disease.
Aortic stenosis and regurgitation.
Mitral stenosis and regurgitation.
Angina pectoris.

---

## CASE 12172

CHILDREN'S MEDICAL DEPARTMENT

A five-year-old boy entered December 10. The chief complaints were swelling of both sides of the neck and of the axillae and groins, malaise, possible fever, and apathy of two weeks' duration.

The family history was good. His past history was also good, except for an attack of scarlet fever two years before admission. He had always been strong and well. He had had no sore throats since his tonsils and adenoids were removed sixteen months before admission. His bowels were regular with castoria twice a week.

Two weeks before admission his mother noticed that the boy's neck was considerably swollen on both sides. He also seemed pale and was rather listless. He had complained once or twice of some abdominal pain and vomited twice during the past week.

Examination showed a well nourished, somewhat pale but not very sick looking boy, frightened and crying a great deal. There was bilateral cervical swelling under the ears. The head seemed to be held somewhat rigid, but moved easily. The eyes, ears and nose were negative. The lips showed a few bloody crusts. The gums bled easily. The tonsils were possibly enlarged, with pitted tags. The pharynx was red. The lymph nodes in the submaxillary region on both sides were enlarged, some of them to the size of a hazel nut. They lay close together and were free from the skin. The axillary nodes were smaller. The inguinal nodes were enlarged; the chains could be felt down the thighs. The nodes were hard and distinct from each other, not tender or painful. The heart and lungs were negative. The abdomen was slightly enlarged in the upper half. The liver could be felt three fingerbreadths below the costal margin as a smooth regular enlargement. The rest of the examination was negative.

The amount of urine was not recorded. The urine was cloudy at one of two examinations, specific gravity 1.013 to 1.016, a slight trace of albumin once, a few to rare red and white blood corpuscles at both examinations. The blood showed 535,000 to 610,000 leucocytes, 1-2 per cent. polynuclears, 94 to 93 per cent. questionable immature lymphocytes, hemoglobin 90 to 75 per cent., reds 5,130,000 to 3,800,000. There were very few platelets. The fragility of the red cells began at 46, was complete at 22. The coagulation time was 6 minutes by the capillary method. The bleeding time was much prolonged, 20 minutes to 2 hours and 40 minutes, depending on the puncture. A Wassermann was negative.

X-ray showed the supracardiac dullness definitely increased along the right border of the sternum. The shape of the shadow suggested enlarged thymus. There was also some increase in the hilus shadows. No definitely calcified glands were seen. The lung fields were clear. The plate was taken at full expiration. Another plate at full inspiration was asked for.

The temperature was 98.1° to 101.5°, the pulse 62 to 40, the respiration 20 to 54.

The child looked very ill, and paler than the

first report of the hemoglobin and red count would account for. The day after admission the neck seemed more swollen. It measured 16¼ inches in a line over the upper lip and the greatest glandular enlargement. The tongue and the soft palate showed hemorrhagic spots which did not fade on pressure. The abdomen and hands showed blue spots of various sizes. The mediastinal dullness seemed to be slightly greater over the sternal region. Vertebral dullness extended as far down as the fourth dorsal. After an ear puncture which bled for three hours there remained a large hemorrhagic ecchymosis in each ear lobe.

The night of December 12 the child vomited three times, sour vomitus with milk curd. He was in a cold sweat, with slow and weak pulse. The heart action was slow, with some prolonged pauses. The pupils were contracted, but reacted to light. The systolic blood pressure was 96, the diastolic 70.

At three in the morning he was in bilateral clonic convulsions involving the flexor and abductor muscles of both upper and lower extremities. The knee-jerks were much exaggerated. No Babinski was elicited. The entire body was spastic. Between the convulsions the body was in a hypertonic state, the hands, feet and mouth held rigid. He vomited dark material which did not resemble blood. The pupils were much contracted, and reacted to light very sluggishly if at all. The pulse was very slow, 50, and at times irregular. The temperature was subnormal, 97°. The skin was cold and clammy. The blood pressure was 90 systolic, 70 diastolic. Early that morning, December 13, he died.

## Discussion

BY ARTHUR BATES LYON, M.D.

The rather sudden and nearly simultaneous swelling of the peripheral lymph nodes in neck, axillae and groins leads at once to the suspicion of malignant disease. This potentiality is proved beyond doubt by the examination of the blood. A white count running into six figures means one thing and only one; that is leukemia. This is lymphatic leukemia, as the differential count shows. The rapid evolution of symptoms shows it to be acute.

Fortunately leukemia is not a common disease in children, for there is no recovery. The acute lymphatic form is the least rare, and is said to appear more often in boys than in girls.

The physical examination does not advance us much in our knowledge of the case. It gives no lead toward any other condition than the leukemia itself. The hard, non-tender, discrete and variably enlarged lymph nodes are characteristic. So too is the enlarged liver. The spleen—not so large in this type of leukemia as in the splenomyelogenous—ought to have been palpable, though apparently it was not felt. The tendency to bleed is almost always present. The fever goes with the leukemia itself; it is no proof of infection.

The number of blood platelets is always greatly diminished in this disease, and is the reason for the tremendously prolonged bleeding time that was found.

I cannot help feeling that the red blood counts and the hemoglobin estimations were consistently too high. Evidently, from the statement that the boy was paler than the findings accounted for, somebody else had the same suspicion.

If there was, as the X-ray suggests, a persistent thymus gland in this boy, it constitutes an adventitious finding. If you take routine X-ray plates of a series of children of this age you will find a certain number show this phenomenon, regardless of other pathology. There is no necessity to associate it with the leukemia.

The mediastinal and bronchial lymphatic glands may be more enlarged than the X-ray can substantiate; especially as from a roentgenological standpoint they may not be very dense. The maker of the physical examination had such enlargement in mind, I suppose, when he described increased paravertebral dullness as far down as the fourth dorsal. It is hard in such a case not to be influenced by the known pathology present; the diagnosis by physical examination of enlarged bronchial glands is notoriously difficult.

What of the convulsive seizures that ushered in the end? I should say probably hemorrhage, or hemorrhages, into the brain, as he had had more or less generally, visibly or invisibly. Sometimes these patients die of an intercurrent infection, but the record gives no data which warrant our reading one into this picture. I fear we shall have to leave that question to the pathologist.

The boy vomited dark material "which did not resemble blood." But I am suspicious that it was blood, nevertheless.

Why the abnormally slow pulse? I do not know. One might say it was the reflection of intracranial hemorrhage, but it was recorded before any signs of brain involvement were noted. Nor is the blood pressure as high as one might expect from much bleeding in the brain.

### SUMMARY

We ought to find the direct evidence of lymphatic leukemia. There will be a general hyperplasia of the lymph nodes. The liver will be smoothly enlarged from agglomerations of lymphoid tissue which it contains; possibly to some extent the spleen; maybe other organs. The bone marrow will be paler than normal. All the organs will show the anemia secondary to the disease and to its symptomatic bleeding. There will be multiple areas of hemorrhage in

many viscera, including the brain. There may be a persistent enlarged thymus. If there be other pathology, there is little to indicate it.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Acute lymphatic leukemia.
Cerebral hemorrhage.

DR. ARTHUR BATES LYON'S DIAGNOSIS

Acute lymphatic leukemia. (Enlarged lymph nodes, liver and spleen.)
Multiple areas of hemorrhage in many viscera, including the brain.
Possibly enlarged thymus.

ANATOMICAL DIAGNOSIS

Lymphatic leukemia.
Leukemic infiltration of the spleen, liver, kidneys, bone marrow, lungs, thymus, and various lymph glands.
Hypertrophy of the spleen and the liver.
Purpura hemorrhagica.
Cerebral hemorrhage.

DR. RICHARDSON: Head: The inner surface of the scalp at one place showed a hemorrhagic area about 2 cm. across. The meninges were negative. The pial vessels of the convexities were flat and rather empty. The sinuses were rather empty. The brain weighed 1380 grams. The tissue generally was rather pale and dry. In the region of the right frontal lobe, occupying the greater part of the inferior two-thirds, there was a mass of disintegrated brain tissue and much blood clot and blood 6 cm. in dimensions. The lower margin of the hemorrhage extended to the region of the inferior cortex, which broke on manipulation. Posteriorly the hemorrhage had ruptured into the right lateral ventricle and into the region of the anterior border of the caudate nucleus. This nucleus in the region of its anterior portion was involved by the hemorrhage for a short distance into its substance. The ventricles contained considerable blood clot and blood. The brain tissue margining the area of hemorrhage showed numerous minute to small hemorrhagic areas, and these areas became confluent and disappeared in the great hemorrhagic area. In the anterior portion of the right occipital lobe just below the superior cortex there was a small discrete area of hemorrhage 1 cm. across. In the anterior portion of the pons just below the posterior surface there was another small discrete area of hemorrhage 7 mm. across. No hemorrhagic areas were found elsewhere.

Trunk: The cervical glands presented as a mass rounding up under the skin just below the jaw on each side, and there were enlarged gland masses in the axillary and inguinal regions on each side. These enlarged glands measured up to 3 cm. across. The lobes of the ears showed purplish areas where they were tapped. The cutaneous vessels of the anterior thoracic wall were slightly prominent. The skin of the hands and upper extremities showed scattered small purplish hemorrhagic areas, and there were some over the lower extremities, and a few minute to small areas over the lower abdominal wall, the scrotum, and the root of the penis. In the left cubital space there were brown-red spots and purplish areas. The lower half of the abdomen showed no definite distension, but the upper half bulged anteriorly and laterally. There was subcutaneous fat in small amount. The muscles were negative except that there was a hemorrhagic area in the right pectoral muscle about 3 cm. across.

The peritoneal cavity was moist. The peritoneum over the intestines showed scattered small purplish hemorrhagic areas.

Small intestine. The Peyer's patches were well marked and showed in instances small hemorrhagic areas.

Large intestine. The mucosa showed a few scattered hemorrhagic areas.

The mesenteric glands were generally enlarged and presented as discrete and confluent masses. The glands measured from a few mm. up to 3 cm. across. The retroperitoneal glands generally were enlarged and presented as discrete and confluent masses up to 4 cm. across. The tissue of the various enlarged glands was plump, softer and firmer, and pale to purplish brown-red. The section surfaces showed small hemorrhagic areas. The anterior margin of the liver was 9 cm. below the costal border in the right mammillary line. The lower border of the lower lobe of the spleen was 7 cm. below the costal border in the left mammillary line.

The pleural cavities contained a very small amount of thin pale reddish fairly clear fluid. Pleural adhesions. Right, none. Left, a small band to the diaphragm.

The thyroid was negative. The thymus gland weighed 25 grams,—moderately enlarged. The tissue was plump, rather meaty, and purplish brown-red with lighter areas here and there. The bronchial glands were enlarged up to 2½ cm. across. They were best marked along the trachea and the region of the thymus. The tissue was like that of the other enlarged glands.

The section surfaces of the lungs in places showed small hemorrhagic areas.

The blood in the heart and vessels was dark red in color and muddy.

The liver weighed 1050 grams,—moderately enlarged. The surface was smooth. The tissue was plump, of good consistence, pale purplish brown-red and homogeneous. No definite mottling was made out.

The spleen weighed 400 grams,—much enlarged. The surface was smooth. The tissue was plump purplish brown-red, homogeneous, with pale prominent follicles.

The kidneys combined weighed 150 grams. The capsules stripped. The surfaces were pinkish brown-red and smooth. The tissue was of good consistence, rather wet, and pinkish brown-red. No definite mottling made out. The markings were visible. The cortex measured 3 mm. The mucosa of the pelves showed scattered small hemorrhagic areas. The ureters were negative. The bladder was distended with pale fairly clear urine. The mucosa showed scattered over it minute to small hemorrhagic areas. Testes: In the section surfaces there were a few minute to small hemorrhagic areas.

The bone marrow of the right femur was dark brown-red, rather mushy and abundant. The marrow of the lumbar vertebrae was dark brownish-red, as was the marrow of the ribs.

A case then of lymphatic leukemia with leukemic infiltration of the various organs, bone marrow, thymus and various lymph glands, with purpura hemorrhagica and cerebral hemorrhage.

DR. LYON: Dr. Richardson, there is one question that interests me, although in a case like this it may be of more academic than practical importance. That is, the use of the term purpura hemorrhagica. There are the symptomatic purpuras, we are taught, and the so-called idiopathic. The purpura in this case I have regarded as purely symptomatic, as part and parcel of the leukemia itself, and as such hardly entitled to a separate name. But in the anatomical diagnosis you use both the diagnoses leukemia and purpura hemorrhagica. What I am trying to get at is this. Do you think that the purpura here is a symptom of the leukemia, or does it occur by reason of other causes, known or unknown? In other words, have we two conditions simultaneously present, or one symptomatic of another?

DR. RICHARDSON: This case is a leukemia with associated purpura. The question as to the cause of the purpura depends on what view you take. If you take the view of primary purpura and secondary purpura, then this case would be secondary purpura. On the other hand you may group all the purpuras under one general heading and feel that the etiology is obscure.

DR. LYON: About the thymus—that its enlargement was an adventitious finding, I am evidently wrong. It was enlarged as the result of the leukemia, it seems. Is the thymus often involved in leukemia?

DR. RICHARDSON: In many cases no, because there is no thymus. In children I do not know the statistics. We have not had enough cases here to answer that question.

## CASE 12173

### SURGICAL DEPARTMENT

A married Italian woman thirty-six years old entered September 8 complaining of weakness and fever. The history was obtained through an interpreter.

*Family history* unimportant.

*Past history* negative.

*Present illness.* She was perfectly well until five weeks before admission except for a firm swelling in the left upper quadrant which was noticed fifteen years ago. Five weeks ago she felt nauseated and had generalized abdominal pain. She took castor oil. Her bowels moved eight times. After this the pain was better and the next day it was gone. Two days later when she tried to get out of bed she was so weak that she went back to bed and remained there. Three days after the onset of the symptoms her eyes and skin were yellow, her urine was dark red, and she had marked loss of appetite, malaise, and about once in three days fever, sometimes 103°. The jaundice became less and in three weeks disappeared. At the end of the next week she had a miscarriage estimated to be at two months. She had had no pain in the region of the abdominal mass, and it had not undergone any change so far as she knew. For two weeks she had had sore burning micturition and her urine had been cloudy. During the past week she had had occasional cough. She thought she had lost some weight. She felt that she was improving.

*Examination* showed a poorly developed and poorly nourished woman with sallow skin, showing evidence of loss of weight. The sclerae were slightly icteric. Many carious teeth. Pyorrhea. Small adenomatous nodule at the left lobe of the thyroid. Epitrochlear glands palpable. In the left epigastric region an ovoid mass the size of a grapefruit, apparently solid, with irregular surface, moving with respiration but not movable over the deep tissues. Tympanitic note over it. Vaginal examination: Introitus relaxed, small. Bilateral laceration of cervix. Uterus in third degree retroversion, apparently of normal size. Rectal examination confirmed the vaginal. Pupils equal. Right reacted to light through a small excursion, left more promptly. Reflexes normal.

*Laboratory data* before operation. Urine turbid at one of two examinations, specific gravity 1.020-1.022. Both specimens showed bile. Blood: Leucocytes 8,000, polynuclears 82 per cent., hemoglobin 50 per cent., reds 3,040,000, marked achromia. Wassermann negative. Clot retraction 6-12 minutes. Bleeding time 2 minutes.

*X-ray.* Right kidney sharply defined. Left a little indistinct. Both appeared low. There were no abnormal shadows present.—Barium meal: Diaphragm high, apparently fixed on the right side, its outline irregular. There was a dense mass occupying the greater part of the right abdomen, displacing the stomach downward and to the left. There was no abnormality of gastric outline or peristalsis and no gastric

stasis. The head of the barium column had not entered the cecum at six hours and at twenty-four hours was in the rectum. The colon was also low, apparently displaced downward by the mass in the upper quadrant.—No definite pathology in the lungs as far as could be determined from a rather poor plate.

Before operation the temperature was 97.5° to 102.2°, the pulse 80 to 100, the respiration normal.

September 12 operation was done. During it the patient's condition became very poor. She was given 2000 c.c. of saline intravenously and in the ward was put in shock position and given a subpectoral. She reacted well. By evening the pulse was of good quality and had fallen from 120 to 100. There was profuse drainage from the wound. Although she continued to run a rather high temperature, by the 20th she was in entirely satisfactory condition, taking nourishment without nausea or vomiting. The night of September 21 the temperature rose to 102° with no obvious cause. A sinus tract about 8 cm. deep was open draining a small amount. She continued to show a persistent anemia. October 11 there was discharge of bile which saturated the dressing. The pulse was 120, the temperature 102°. There was no sign of hemorrhage. Examination showed an enormous liver reaching almost to the iliac crest, smooth and non-tender. She complained of abdominal pain which soon disappeared. There was perhaps extremely slight jaundice of the sclerae. Two days later the liver edge was not palpable. There was continuous bile discharge. During the next four days there was marked decrease in the discharge. The patient was comfortable. The temperature varied slightly above normal. Bile was present in the stools. Between October 23 and 29 three deep X-ray treatments were given over the liver.

There was practically no change in the condition until November 6, when the temperature rose to 104° and the patient had slight cough. November 8 some signs of consolidation in the lower lobe of the lung had appeared over night. During the next three days the lung signs decreased and the temperature fell. A medical consultant suggested the possibility of infarct and advised X-ray to settle the question as to something below the right diaphragm. X-ray showed the right diaphragm apparently higher than in the previous examination. There was absence of respiratory excursion on this side. The outline of the diaphragm was very indistinct, and the costophrenic sinus practically obliterated. The diaphragm was somewhat limited on the left. On the right above the diaphragm there was a narrow band of increased density extending across the chest, probably representing a pathological process in the lung. The heart was not displaced. The left lung field was clear. During the past month however the general condition had shown no improvement.

November 14 in preparation for operation the patient was given one-sixth of a grain of morphia and one two-hundredths of a grain of scopolamin at nine o'clock. At ten o'clock the dose was repeated. Fifteen minutes later her face was flushed, her pupils were dilated, and she coughed up 250 c.c. of bile containing mucus. When next seen she was in the amphitheatre awaiting operation. She was in profound coma, her eyes were rolled up, her pupils dilated. She was still coughing up considerable amounts of bile-stained material. She was sent to the ward and given caffein and oxygen. She was pulseless at the wrist. The respirations were shallow and death seemed only a few moments away. After the caffein the pulse and respiration improved and in about ten minutes she sat up in bed. Her condition improved rapidly and was as good as before. Two days later it was better than before the 14th. Operation was postponed and postural drainage tried. This yielded about 60 c.c. of sputum twice November 17. There was no bile present grossly. November 18 films were not entirely satisfactory but seemed to show the previously described area of increased density in the lower right chest to have increased in extent. The diaphragm could not be definitely visualized. November 19 a chest tap in the sixth space, midaxillary line, gave 15 c.c. of yellowish purulent fluid.

November 21 a second operation was done. The later history is given with the operation.

## Discussion

BY RICHARD H. MILLER, M.D.

The striking thing about the mass was its irregularity. I remember it very distinctly. There were no other masses to be felt in the liver—just this one large tumor.

The bleeding time is interpreted to mean that there was no particular delay in clotting and no increase in time of bleeding. It was done in the presence of jaundice, simply to determine whether she would bleed more than she ought to.

At physical examination no dense mass was obvious on the right side. The mass was more on the left.

After a few days in the hospital she was operated on.

Dr. Edward L. Young: Did you do an echinococcus fixation test?

Dr. Miller: Yes, we did, and the echinococcus fixation test was positive. The thing which was outstanding in this case was the fact that this tumor which the description says was tympanitic—I do not remember the tympany over it—when tapped with the fingers gave the most marked and distinct thrill that one could

possibly get, and that was the typical thrill which is described as existing in echinococcus cyst,—a big cyst full of daughter cysts.

Now that we have the diagnosis I may say that the report of the blood smear was left out of this summary intentionally. It did not show any eosinophilia. There should be eosinophilia in this case, and it was not present.

There are two or three points which are interesting and pretty hard to explain. In the first place, why should she go for fifteen years with an echinococcus cyst in the liver and then suddenly flare up in this way with temperature and jaundice? I think the answer to that must be that there was a temporary infectious process. Whether or not it started in the cyst I do not know. I think it did not. But there must have been an infectious jaundice due to an infectious cholangitis, that is, an infection of the bile-ducts. There is no other way in which one can easily account for that. She certainly had no gall-stones and no cholecystitis.

DR. YOUNG: I think they often do show that. I remember one echinococcus cyst that came in for acute cholecystitis. I wondered whether through a slight rupture or change in the cyst there was enough leakage of what is said to be irritating fluid to stir up an infectious process.

DR. MILLER: At any rate the miscarriage had nothing to do with it and was merely coincidental. The urinary symptoms as shown by burning micturition and cloudy urine were evidently a purely secondary matter and not of primary importance. There was nothing in the physical examination requiring any special comment.

The leucocyte count of 8,000 previous to operation demonstrates that there was no marked septic process going on at that time. She was anemic of course, as shown by the hemoglobin, the red cells, and the achromia, but I think that could naturally accompany such a condition, which had gone on all this time.

On the strength of this mass in the left epigastrium which had a definite thrill we felt warranted in making a diagnosis of echinococcus cyst. We ruled out the two most common lesions: (1) gumma, because we had a negative Wassermann and because of the irregularity of the mass and the thrill; (2) cancer, because there was no history suggestive of cancer, and cancer of the liver does not ordinarily occur in one huge mass like this, but in lumps and nodules scattered all through the liver. Furthermore, in a case of cancer we should have been able to find suggestions, at least, of a primary focus. Any other infection would probably not give rise to a large tumor without symptoms,—any other infection such as tuberculosis. So we had a pretty definite diagnosis of echinococcus cyst.

A long incision was made on the left side of the epigastrium, and immediately there presented into the wound an enormous irregular tumor arising from the under surface of the liver, adherent to the stomach, the pancreas and somewhat to the overlying omentum. The question came up at once whether to bring it up into the wound, suture it, and then scrape it out, which is frequently done with this type of tumor and is called "marsupializing," or whether to take it out. It seemed at the time that this was hanging on the under surface of the liver, and there seemed to be a perfectly good possibility of taking the whole thing out.

PATHOLOGICAL REPORT SEPTEMBER 12

Echinococcus fixation test strongly positive.

PRE-OPERATIVE DIAGNOSIS SEPTEMBER 12

Echinococcus cyst of liver.

FIRST OPERATION

Gas-ether. Seven inch incision in epigastrium to the left of the midline, with an additional incision two inches long at a right angle to this under the border of the ribs. An irregular cystic mass measuring about 12 x 12 x 10 cm. was found arising from the under surface of the left lobe of the liver, extending up as high as the diaphragm, adherent to the pancreas posteriorly, to the stomach below, and to the anterior abdominal wall in front. At first it seemed impossible to remove this, but it was decided that an attempt ought to be made. All adhesions were rapidly clamped and cut, and the operation, which at first seemed difficult, became very easy. The cyst was pulled off from the under surface of the liver without difficulty and with practically no bleeding. In the end the delivery was simple and it was removed entire, although it ruptured in one place during removal. One rubber tissue wick.

PATHOLOGICAL REPORT

A cystic tumor the size of a cantaloupe, with thick walls. It is filled with daughter cysts and lined by a white chitinous membrane.

Tenia echinococcus.

FURTHER DISCUSSION

The first incision was obviously not enough to allow us to get through, so another was made at a right angle to it. The mass was estimated to be twelve by twelve by ten cm., but it was certainly very much larger that that. There was no marked bleeding and no obvious trouble, although the patient was in rather poor condition from the shock. On taking it out the tumor ruptured in one place and was found to be full of little cysts which reminded one of soap-bubbles. A few escaped into the abdominal cavity, but not very many.

After operation there was serous drainage, not very much bile.

We never could find out just why this discharge of bile on October 11 happened. There may have been a steady leakage of bile encapsulated by adhesions which may have broken loose and come out through the incision.

The liver at this time seemed to have descended to the iliac crest. Two days later it was not palpable, and I do not know what happened.

DR. YOUNG: Isn't that a very sudden change?

DR. MILLER: It is, and it makes one question the accuracy of the observation. I did not see it myself, but I think the house officers were perfectly certain of what they thought was their observation. I do not understand how the liver could have increased to that size and then gone down.

X-ray treatment has been thought to be of some avail in echinococcus cyst. Have you had enough cases, Dr. Holmes, to be able to judge about it?

DR. HOLMES: We have had very few cases that have had X-ray treatment alone. It has always been combined with surgery. There is however very good evidence that we can kill the echinococcus organism by radiation, and my contention is that we should kill these before operation. Then in removing the cyst they would not start to grow, if any were spilled out.

DR. MILLER: She went on for a month without any marked improvement, with signs suggesting trouble on the right side, and we could not make a diagnosis as to whether it was subdiaphragmatic abscess or something above the diaphragm. We thought of a possible extension of the process through from underneath, involving the base on the right, with possibly empyema, because it seemed like a septic process.

DR. HOLMES: This is the first plate, taken on October 1, and shows the high diaphragm on the right. In this particular plate the outline of the diaphragm is rather sharp and there is no evidence of anything in the chest at this time. So that when we take the whole series we have good evidence from the X-ray that the process began below the diaphragm. The second plate, taken in November, shows what the other did, and also a beginning haziness above the diaphragm and a partial occlusion of the costohepatic angle, so that there is now evidence of something affecting the lung. The later plates show simply gradual progression of the process.

DR. MILLER: The question came up, and it was a very important question, whether there was pus below the diaphragm or above it. Could you state from this plate?

DR. HOLMES: In the first plate I could state that there was something below,—it might have been pus or a tumor. In the second set I could state that there was not enough in the chest to account for the high position of the diaphragm, that she had something in the chest which was probably pus, and probably pus below the diaphragm. The echinococcus would not extend through the diaphragm in that way. This is probably an abscess.

DR. MILLER: It was felt that the proper thing to do was to explore that region with the idea of locating probably a subdiaphragmatic abscess, or if not, an abscess low down in the pleural cavity. We thought she could not stand ether, so she was given the scopolamin and morphine. I went in to see her as she was awaiting operation and I thought she was dying, as near dead as we often see. Her pupils were dilated, she was unconscious, she had the pallor of death, and she had been vomiting bile-stained fluid with mucus in it. It looked like a mixture of bile and pus. She rapidly improved under stimulation. During the following days she drained through the lungs a lot of this bile-colored thick fluid, so that we tried postural drainage as one would with a lung abscess. This obviously did not come from the stomach, it was not vomited bile, it was coughed up, and the only conclusion we could reach was that an accumulation of bile and pus below the diaphragm had perforated up through the diaphragm and established communication with some of the small bronchi, and that this fluid was coming up through the lungs.

PRE-OPERATIVE DIAGNOSIS NOVEMBER 21

Empyema.

SECOND OPERATION

Novocain infiltration. A needle was introduced into the sixth space in the midaxillary line on the right. Pus was obtained. A small incision was then made in the skin and a trocar introduced through which a catheter was inserted. On aspiration a thick semi-purulent fluid containing many collapsed cysts was obtained. It was evident that proper drainage could not be obtained through the catheter, so a portion of the seventh rib was resected and a large cavity apparently below the diaphragm was opened into. A considerable amount of pus and many cysts were removed. One rubber tube inserted for drainage. The pleural cavity was not opened.

PATHOLOGICAL REPORT

Irregular fragments of small cysts some of which show smooth white chitinous membrane.

Tenia echinococcus.

FURTHER DISCUSSION

We wanted to do as little as we could. Through the trocar a catheter was inserted, a big syringe attached, and by that we sucked out some cysts. It became obvious that since we had to suck as hard as possible to get them out they would not drain through the catheter. A rib was resected and a large cavity was opened into which contained a lot of this bile fluid and was full of cysts. So far as we could determine the cavity was below the diaphragm,

which had been pushed up, and so far as I recall there was no obvious interchange of air as if there were a bronchial fistula through that abscess.

That is the end of the clinical story. I will now give very briefly what I know of the later history. She recovered perfectly well. The original incision on the left over the cyst healed. The incision into the chest for the subdiaphragmatic abscess healed, and she went out of the hospital fairly well.

Dr. Wright examined the cysts and said there was no way of telling whether they were alive or not.

A SURGEON: Is there any evidence that those cysts had been multiplying?

DR. MILLER: No, there is not. The only thing is that before the original operation she had no signs of trouble on the right under the diaphragm. Now she either had a cyst which grew rapidly, or she had an old cyst which we did not recognize, which became infected either as a result of or coincident with the first operation.

DR. HOLMES: The evidence then is rather against those cysts being killed by radiation.

DR. MILLER: Yes and no. I may say I do not believe they grew. I think there was another cyst in the right lobe which got infected. Dr. Churchill, from the point of view of the thoracic surgeon, it is pretty unusual, isn't it, to have this sequence of events?

DR. EDWARD D. CHURCHILL: I think, as Dr. Holmes pointed out, it is unusual for an echinococcus cyst of the liver to rupture through the diaphragm into the lung, although such cases have been reported, particularly from South America. I wonder whether it was a second cyst or a residual subdiaphragmatic abscess which broke through. It does not seem probable that cysts got from the field of your operation to the subphrenic space.

DR. MILLER: Yes. I think there must have been another big cyst with daughter cysts on the right. But I was particularly interested in the question of that getting through and perforating into a bronchus so that she could cough up a lot. Isn't that pretty unusual?

DR. CHURCHILL: Not for a subdiaphragmatic abscess or for a cyst which is secondarily infected.

DR. HOLMES: How about an echinococcus cyst in the lung? If there are two eggs one may go into the liver, the other into the lungs.

DR. CHURCHILL: The thing that struck me was that coincidentally with the rupture of both cysts the patient went into deep shock, once on the table and again when the abscess ruptured. Have you considered the possibility of that being an anaphylactic reaction?

Spilling of the cystic fluid at operation is known to bring on anaphylactic shock. I should think equally, the sudden rupture of a cyst into the bronchial tree might bring on this phenomenon.

DR. MILLER: I think it might be, although we had not actually considered that, because at the first operation when she suddenly went into collapse the fluid had not been spilled around enough, and the operation was enough to send anyone into shock. In the second place, we considered that there was scopolamin poisoning, and we considered it one of the lessons taught by this case that we cannot use scopolamin without great care. But your suggestion is perfectly sound and good. Dr. Richardson, have you anything to add to this?

DR. OSCAR RICHARDSON: It interested me. I thought it was probably a cyst up there. Was the original cyst infected? Did you get pus?

DR. MILLER: At the second operation, not at the first.

DR. RICHARDSON: You feel perfectly sure that you did not have a cyst in the lung?

DR. MILLER: I do not think we had.

She came in about a year afterwards with an acute gangrenous perforated appendix. Dr. McIver operated on her at that time, and I will ask Dr. McIver to tell what he found.

DR. MONROE A. MCIVER: The history and physical findings seemed to be perfectly consistent with an attack of appendicitis, and on abdominal exploration an acute gangrenous appendicitis was found. In the neighborhood of the ileocecal angle there were one or two small gelatinous or slightly translucent cysts which I thought, in view of this previous history, probably represented transplanted cysts. I did not do anything about them. It would have involved more dissecting than I cared to do under the conditions.

DR. MILLER: I might add that she came in here and had a curettage for chronic endometritis last fall, and the pathological examination of that was unimportant.

DR. MCIVER: I saw this patient a few weeks ago in the Out-Patient Department. Her general appearance had improved so much that I did not recognize her. She had put on I should think twenty or thirty pounds and seemed perfectly well. On palpation of the abdomen I could not feel the liver edge or any masses.

DR. RICHARDSON: You did not find any cysts in the appendix?

DR. MCIVER: I did not. At the time of the operation the appendix was quite gangrenous.

DIAGNOSIS

First Admission

Echinococcus cyst of the liver.
Subphrenic abscess.

Second Admission

Acute gangrenous appendix.

Third Admission

Menorrhagia.
Hyperplastic endometritis.

THE BOSTON

# Medical and Surgical Journal

*Established in 1828*

Published by The Massachusetts Medical Society under the jurisdiction of the following-named committee:

For three years — William H. Robey, Jr., M.D.; Roger I. Lee, M.D.; Robert B. Osgood, M.D.

For two years — John W. Bartol, M.D.; Horace D. Arnold, M.D.; Channing Frothingham, M.D.

For one year — Homer Gage, M.D., *Chairman*; Edward C. Streeter, M.D.; Edward W. Taylor, M.D.

Editorial Staff

David L. Edsall, M.D.
Walter B. Cannon, M.D.
Reid Hunt, M.D.
Francis W. Peabody, M.D.
John P. Sutherland, M.D.
S. Burt Wolbach, M.D.
George R. Minot, M.D.
Frank H. Lahey, M.D.
Stephen Rushmore, M.D.

Walter P. Bowers, M.D., *Managing Editor*

Associate Editors

George G. Smith, M.D.
William B. Breed, M.D.
Joseph Garland, M.D.

Subscription Terms: *$6.00 per year in advance, postage paid for the United States, $7.56 per year for all foreign countries belonging to the Postal Union.*

*Material for early publication should be received not later than noon on Saturday. Orders for reprints must be sent to the Journal office, 126 Massachusetts Ave.*

*The Journal does not hold itself responsible for statements made by any contributor.*

*Communications should be addressed to The Boston Medical and Surgical Journal, 126 Massachusetts Ave., Boston, Mass.*

## THE MASSACHUSETTS MEDICAL BENEVOLENT SOCIETY

From the fact that of the more than four thousand members of the Massachusetts Medical Society only about two hundred belong to the Massachusetts Medical Benevolent Society, it would seem that the objects and the work of the latter were not as generally known as they should be. Doubtless this is partly due to the fact that the annual reports of the Society are not published and hence do not receive the publicity of many of our medical societies.

The Massachusetts Medical Benevolent Society was organized by a few members of the parent society in 1857 for the purpose of giving aid to destitute physicians and their families. The Society was incorporated in 1871 to enable it to hold property, real and personal, which it is now authorized to do to the extent of one hundred and fifty thousand dollars. It is entirely independent of the parent society.

The Society is composed of members of the Massachusetts Medical Society. The officers are a President, Vice-President, Treasurer, Secretary and nine Trustees. The first four officers and three of the Trustees are elected annually on the last Thursday in October. These thirteen officers constitute the Council of the Society and have charge of the funds and the distribution of the charities of the Society.

Persons desiring membership are nominated to the Council through the Secretary and confirmed by the Society. Admission fee is five dollars and the annual dues are two dollars for twenty years when members are excused from all further assessments. The payment of twenty-five dollars at any one time makes one a life member and excuses him from subsequent assessments. "The donation of fifty dollars or more at one time shall entitle the donor to be inscribed among the Benefactors of the Society."

For sixty-nine years this Society has been giving aid to those needy physicians and their families who are or have been residents of this Commonwealth. Usually the aid is in the form of annuities from one to three hundred dollars, payable quarterly. Furthermore, "The President and Treasurer shall have authority to render temporary assistance to any needy members of the medical profession, or to their widows and children." Practically all applicants have received assistance of some sort. There is no waiting list and, so far as the writer knows, there never has been one.

According to the last annual report the funds of the Society amount to $74,800.00. The income for the year was $7,369.73 and the distribution amounted to $5,569.52. It should be said that three thousand dollars of the annual income is a perpetual annuity secured through the enterprise and influence of the Treasurer from an outside source. There are no overhead expenses in the way of up-keep of real estate, taxes, rents, interest, commissions, etc. The entire income is free of all incumbrances.

At present there are nineteen annuitants upon the list; five physicians and nine widows and five daughters of physicians. The affairs of this Society have been most admirably managed for thirty-five years by the present Treasurer, Dr. W. L. Richardson, to whom, alone, is due much of the success of this worthy charity. Dr. George W. Gay is President.

The annuities although individually small have contributed to the comfort of many beneficiaries, but it may be that larger annuities would be appreciated in many instances and it is possible that the needs of some worthy doctors have not been brought to the attention of the Society. We respectfully suggest that all who may be able should send contributions to the Treasurer and that suggestions for new members should be referred to the Secretary, Dr. Robert M. Green. This Society should have a much larger membership list.

## DELAYED JUSTICE

In the *Evening Transcript* of April 9th was a long account from London of the death of Dr. F. W. Axham who in 1912 was deprived of

his license by the Royal College of Physicians of Edinburgh. His offence had been that as anaesthetist he had assisted an osteopath, Herbert Barker, who was later knighted for his services.

On January 19, 1926, the College voted to remove the suspension and return the diploma to Dr. Axham. Unfortunately he died before the General Medical Council had taken final action in the matter.

Thus for a moment is revived the memory of an incident which attracted considerable attention at the time and which permitted George Bernard Shaw to vent his sarcasm on the medical profession.

Fortunately such occurrences as this become less common as knowledge and tolerance increase. It may be a little comfort to realize that much worse sins have been committed in the name of Christianity than in the maintenance of medical ethics. The futility of such ostracism is obvious, because the public does not understand and will not sympathize with the objects of those who attempt thus to maintain professional standards. The public considers the martyrdom of a man who embraces new ideas. No matter how false these ideas may be they gain ground as they could in no other way.

Education and time are the only remedies which overcome false doctrines. Fraud, however, cannot be treated so leniently.

Medical organizations are called upon to deal with questions of ethics which are not strictly subject to the provision of the statutes and endeavor to analyze motives and effects with the purpose of administering justice without doing harm to individuals or society at large. All differences of opinion should be given generous consideration.

Criminal acts or gross departure from moral standards must be dealt with by Boards or officials clothed with more definite authority. In this Commonwealth we are perhaps too much inclined to depend on the legal authorities.

It is the difficult task of the Board of Registration in Medicine to check dishonesty without injustice to the honest fanatic and yet prevent harm to the public through the unwise activities of mistaken enthusiasts. In the decisions of the Board sectarianism has no place. Adequate knowledge and the honest conscientious application of that knowledge are the sole criteria. One standard of fitness for all is the only safe basis for registration.

## THE WATER SUPPLY FOR METROPOLITAN BOSTON

With regard to the technical problems involved in securing an adequate water supply for the Metropolitan District physicians have no special knowledge. It must, however, be a source of gratification to the medical profession of the Commonwealth to note that under the stimulus of a Governor deeply interested in public health matters the eminent engineers who have presented various solutions of the problems involved finally agree essentially on the plan outlined by Mr. Goodnough, the engineer of the Department of Public Health. The highest credit must be given to him and to Dr. Henry P. Walcott, the Chairman of the older State Board of Health and later of the Metropolitan Water Commission, for their devoted and able services. After years of controversy their wisdom, foresight and sound judgment are confirmed by the leading engineers of the country.

## THIS WEEK'S ISSUE

Contains articles by the following named authors:

Dameshek, William, M.D. Harvard Medical School 1923; Junior Visiting Physician, Boston Dispensary; Teaching Assistant, Boston City Hospital; Teaching Assistant, Tufts Medical School. His subject is "The Reticulated Red Cells—Their Clinical Significance," page 759. His address is 484 Commonwealth Ave., Boston.

Thoma, Kurt H., D.M.D. Harvard University Dental School; Assistant Professor of Oral Pathology, Harvard University Dental School; Member of Research Committee, Harvard University Dental School; Visiting Aural Surgeon, Long Island Hospital, etc. His subject is "A Review of Our Present Knowledge of the Pathology and Roentgen Diagnosis of Periapical Tooth Infection," page 768. His address is 43 Bay State Road, Boston.

Seham, Max, M.D. University of Minnesota Medical School 1910; Assistant Professor of Pediatrics, University of Minnesota Medical School. His subject is "Chronic Fatigue in the School Child," page 770. His address is 2200 S. Fremont Ave., Minneapolis, Minn.

Jones, T. Duckett, A.B. Virginia Military Institute 1919; M.D. University of Virginia Medical School 1923; Resident in Cardiology, Massachusetts General Hospital. Associated with him is

Sprague, Howard B.; A.B., M.D. Harvard Medical School 1922; Assistant in Medicine, Courses for Graduates, Harvard Medical School; Graduate Assistant in Medicine, Massachusetts General Hospital; Visiting Physician, House of the Good Samaritan. They write on "Progress in Cardiovascular Disease," page 777. Their address is Massachusetts General Hospital.

## LEGISLATIVE NOTE

HOUSE Bill No. 145 has been passed to be engrossed in House and Senate. It is an act relative to Subsidies to Cities and Towns for Pulmonary Tuberculosis Cases. This Bill reads as follows:

Section seventy-six of chapter one hundred and eleven of the General Laws is hereby amended by inserting after the word "superintendent," in the twentieth line the words:—or medical director,—by striking out, in the twentieth and twenty-first lines, the words "the district health officer of the district where the hospital is situated," and inserting in place thereof the words:—a member of the department designated by the commissioner,—and by inserting after the word "of," the second time it occurs in the twenty-first line the word:—pulmonary,—so as to read as follows:—*Section 76.* Every town placing its patients suffering from tuberculosis in a county, municipal or incorporated tuberculosis hospital in the commonwealth, or in a building or ward set apart for such patients by a county, municipal or incorporated hospital therein, shall be entitled to receive from the commonwealth a subsidy of five dollars a week for each patient who has a legal settlement therein, provided that such patient is unable to pay for his support, and that his kindred bound by law to maintain him are unable to pay for the same; but a town shall not become entitled to this subsidy unless, upon examination authorized by the department, the sputum of such patient be found to contain bacilli of tuberculosis, nor unless the hospital building or ward be approved by it, and it shall not give such approval unless it has by authority of law, or by permission of the hospital, full authority to inspect the same at all times. The department may at any time withdraw its approval. In the case of hospitals having a bed capacity which, in the opinion of the department, is in excess of the number of beds needed for the localities which these institutions serve for patients exhibiting tubercle bacilli in their sputum, the subsidy above provided shall be allowed for such patients not exhibiting tubercle bacilli in their sputum as, in the joint opinion of the superintendent or medical director of the institution and of a member of the department designated by the commissioner, are bona fide cases of pulmonary tuberculosis and have been in the institution more than thirty days.

## MISCELLANY

### UNIVERSAL ANTITOXIN

THE daily papers state that Dr. Arthur Locke, Seymour Coman Fellow of the University of Chicago, Dr. E. F. Hirsch and Miss Edna Ruth Main, working in the laboratories at St. Luke's Hospital, have evolved a theory, which they feel is supported by experiments, and which if applied through a combined serum will cure or materially modify diseases due to bacterial invasions.

The advantage over specific sera lies in the early application of this combination for it will be unnecessary to differentiate the specific bacteria in a given case.

The idea is not particularly new but the creation of a remedy which has general rather than specific power will add tremendously to the physician's armamentarium. If the expectation of these investigators can pass the critical examination of competent observers, a great life saving treatment is at hand. We hope that they are on the right track but must confess a conservative skepticism.

### FRUIT MADE POISONOUS BY SPRAYS

THE people in England are quite concerned about the dangers incident to the use of fruit which has been subjected to spraying by arsenic and lead compounds. Growers of fruit have claimed that rain washes away these poisonous compounds. Some people contend that the arsenic and lead compound makes a sort of paint on the surface of apples, for example, which is not easily removed.

A dealer in England has been convicted of selling apples contaminated to the extent of one tenth of a grain of arsenic per pound. These apples were grown in America.

The arsenical sprays are used by some growers after the apples are well formed and if nature is unable to wash away the poison, a definite danger exists. It is even contended that all of the arsenic is not on the outside of the apples. Perhaps chemistry will evolve a parasiticide which is not harmful to human beings.

## RECENT DEATH

SCHENCK—DR. ELLEN LOUISE EASTMAN SCHENCK, a practitioner of Ashby, Mass., died at a private hospital in Lunenburg, April 19, 1926, at the age of 71.

Dr. Schenck was born in Woburn, May 24, 1855, daughter of Noah Eastman and Martha McClure Eastman. She was educated in the Woburn schools and was graduated from the Boston University School of Medicine in 1881. She began practice in Fitchburg and in 1899 was married to W. Harmann Schenck, who died in 1913. Dr. Schenck then removed to Ashby, remaining there until her fatal illness, when she went to the hospital in Lunenburg.

### MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH

DISEASES REPORTED FOR THE WEEK ENDING APRIL 17, 1926

| | | | |
|---|---|---|---|
| Anterior poliomyelitis | 1 | Pellagra | 1 |
| Chickenpox | 122 | Pneumonia, lobar | 218 |
| Diphtheria | 41 | Scarlet fever | 271 |
| Dog-bite | 2 | Septic sore throat | 1 |
| Encephalitis lethargica | 1 | Smallpox | 4 |
| Epidemic cerebrospinal meningitis | 2 | Suppurative conjunctivitis | 15 |
| German measles | 297 | Syphilis | 58 |
| Gonorrhea | 81 | Trachoma | 1 |
| Influenza | 132 | Trichinosis | 1 |
| Measles | 881 | Tuberculosis, pulmonary | 183 |
| Mumps | 122 | Tuberculosis, other forms | 16 |
| Ophthalmia neonatorum | 28 | Tuberculosis, hilum | 38 |
| | | Typhoid fever | 8 |
| | | Whooping cough | 344 |

## RESUME OF COMMUNICABLE DISEASES REPORTED IN MASSACHUSETTS

### MARCH, 1926

#### GENERAL PREVALENCE

The common communicable diseases which showed an increase over last month were: Diphtheria, German measles, mumps, scarlet fever and whooping cough.

| | Mar., 1926 | Feb., 1926 | Mar., 1925 |
|---|---|---|---|
| Diphtheria | 304 | 273 | 429 |
| German measles | 1,067 | 478 | 1,067 |
| Mumps | 518 | 347 | 420 |
| Scarlet fever | 1,194 | 1,119 | 1,458 |
| Whooping cough | 2,179 | 1,653 | 700 |

#### RARE DISEASES

*Anterior poliomyelitis* was reported from Dudley, 1; Fall River, 1; Haverhill, 1; Southbridge, 2; total, 5.

*Anthrax* was reported from Haverhill, 2; Lynn, 1; Worcester, 2; total, 5.

*Dog-bite requiring anti-rabic treatment* was reported from Beverly, 1; Boston, 1; Everett, 1; Lowell, 3; Medford, 7; Peabody, 1; Quincy, 1; Revere, 1; Winthrop, 1; total, 17.

*Dysentery* was reported from Ipswich, 1; total, 1.

*Encephalitis lethargica* was reported from Adams, 1; Barnstable, 1; Beverly, 1; Boston, 2; Cambridge, 2; Lawrence, 1; Lynn, 1; Peabody, 1; Salem, 2; Worcester, 1; total, 13.

*Epidemic cerebrospinal meningitis* was reported from Boston, 8; Cambridge, 1; Everett, 1; Lynn, 2; Melrose, 1; Milford, 3; Newburyport, 1; Salem, 1; total, 18.

*Hookworm* was reported from Boston, 1; total, 1.

*Malaria* was reported from Peabody, 1; total, 1.

*Septic sore throat* was reported from Beverly, 1; Boston, 1; Fall River, 2; Franklin, 1; Haverhill, 1; Holliston, 1; Lynn, 1; Wakefield, 1; Wellesley, 1; Westfield, 1; total, 11.

*Tetanus* was reported from Boston, 1; Salem, 1; total, 2.

*Trachoma* was reported from Boston, 3; total, 3.

#### DISTRIBUTION

*All Communicable Diseases*

| | Mar., 1926 | Mar., 1925 |
|---|---|---|
| Total cases (all causes) | 15,110 | 10,529 |
| Case rate per 100,000 population | 358.2 | 253.2 |

*Certain Prevalent Diseases*

*Diphtheria*

| | Mar., 1926 | Mar., 1925 |
|---|---|---|
| Total cases | 304 | 429 |
| Case rate per 100,000 population | 7.2 | 10.3 |

Cities and towns noticeably exceeding their median endemic indexes*:

| | | | | | |
|---|---|---|---|---|---|
| Beverly | (1) | 6 | Webster | (0) | 3 |
| Wakefield | (1) | 6 | Holyoke | (5) | 17 |
| Leicester | (0) | 2 | Northampton | (1) | 4 |

*Measles*

| | Mar., 1926 | Mar., 1925 |
|---|---|---|
| Total cases | 5,490 | 2,747 |
| Case rate per 100,000 population | 130.2 | 66.1 |

Cities and towns noticeably exceeding their median endemic indexes*:

| | | | | | |
|---|---|---|---|---|---|
| Attleboro | (0) | 112 | Winthrop | (3) | 21 |
| Chatham | (0) | 24 | Arlington | (7) | 112 |
| Dartmouth | (0) | 15 | Chelmsford | (0) | 67 |
| Fairhaven | (0) | 9 | Concord | (1) | 31 |
| Middleboro | (1) | 8 | Waltham | (6) | 58 |
| New Bedford | (6) | 92 | Watertown | (3) | 21 |
| Taunton | (1) | 27 | Ashburnham | (0) | 54 |
| Avon | (0) | 28 | Auburn | (0) | 11 |
| Braintree | (2) | 16 | Dana | (1) | 61 |
| Bridgewater | (1) | 96 | Fitchburg | (2) | 215 |
| Brockton | (22) | 182 | Harvard | (6) | 23 |
| Brookline | (17) | 41 | Lancaster | (0) | 36 |
| Cohasset | (0) | 22 | Leominster | (1) | 190 |
| Dedham | (1) | 21 | Millbury | (0) | 13 |
| Framingham | (3) | 7 | Southbridge | (0) | 115 |
| Hopkinton | (0) | 96 | Sturbridge | (0) | 8 |
| Marlboro | (4) | 45 | Templeton | (0) | 12 |
| Needham | (1) | 61 | Uxbridge | (0) | 10 |
| Newton | (14) | 392 | Webster | (0) | 13 |
| No. Attleboro | (0) | 15 | Westboro | (0) | 21 |
| Sharon | (0) | 25 | Agawam | (0) | 25 |
| Wellesley | (5) | 57 | Chicopee | (0) | 32 |
| Beverly | (4) | 36 | Springfield | (36) | 798 |
| Danvers | (1) | 14 | W. Springfield | (1) | 25 |
| Melrose | (3) | 19 | Westfield | (5) | 140 |
| Newburyport | (4) | 25 | Northfield | (0) | 5 |
| Peabody | (4) | 24 | | | |

*Scarlet Fever*

| | Mar., 1926 | Mar., 1925 |
|---|---|---|
| Total cases | 1,194 | 1,458 |
| Case rate per 100,000 population | 28.3 | 35.1 |

Cities and towns noticeably exceeding their median endemic indexes*:

| | | | | | |
|---|---|---|---|---|---|
| New Bedford | (18) | 63 | Wakefield | (7) | 15 |
| Ashland | (1) | 5 | Arlington | (4) | 9 |
| Holliston | (0) | 4 | Chelmsford | (0) | 6 |
| Hopkinton | (0) | 3 | Lowell | (19) | 46 |
| Natick | (2) | 6 | Methuen | (3) | 6 |
| Norwood | (2) | 8 | Watertown | (6) | 14 |
| Walpole | (0) | 3 | Winchester | (2) | 7 |
| Amesbury | (3) | 6 | Woburn | (0) | 2 |
| Everett | (19) | 39 | Millbury | (0) | 8 |
| Ipswich | (1) | 6 | Hatfield | (0) | 4 |
| Lynn | (24) | 49 | Palmer | (0) | 2 |
| Stoneham | (1) | 5 | Montague | (0) | 4 |

*Tuberculosis, Pulmonary*

| | Mar., 1926 | Mar., 1925 |
|---|---|---|
| Total cases | 514 | 537 |
| Case rate per 100,000 population | 12.1 | 12.9 |

*Tuberculosis, Other Forms*

| | Mar., 1926 | Mar., 1925 |
|---|---|---|
| Total cases | 103 | 89 |
| Case rate per 100,000 population | 2.4 | 2.1 |

*Typhoid Fever*

| | Mar., 1926 | Mar., 1925 |
|---|---|---|
| Total cases | 20 | 42 |
| Case rate per 100,000 population | .4 | 1.0 |

*Whooping Cough*

| | Mar., 1926 | Mar., 1925 |
|---|---|---|
| Total cases | 2,179 | 700 |
| Case rate per 100,000 population | 51.7 | 16.8 |

Cities and towns noticeably exceeding their median endemic indexes*:

| | | | | | |
|---|---|---|---|---|---|
| Acushnet | (0) | 4 | Wakefield | (0) | 5 |
| Attleboro | (0) | 42 | Arlington | (4) | 60 |
| Dartmouth | (1) | 4 | Belmont | (1) | 56 |
| Taunton | (1) | 8 | Medford | (4) | 46 |
| Westport | (0) | 7 | Somerville | (15) | 39 |
| Avon | (0) | 12 | Waltham | (8) | 57 |
| Boston | (126) | 782 | Watertown | (1) | 24 |
| Braintree | (0) | 5 | Wayland | (0) | 24 |
| Brookline | (8) | 58 | Athol | (0) | 5 |

| | | | | | |
|---|---|---|---|---|---|
| Amesbury | (0) | 6 | Auburn | (0) | 4 |
| Lynn | (12) | 36 | Harvard | (0) | 4 |
| Malden | (12) | 42 | Leominster | (0) | 9 |
| Newburyport | (3) | 18 | Millbury | (0) | 6 |
| Stoneham | (0) | 17 | Northbridge | (0) | 6 |
| Cambridge | (49) | 155 | Upton | (0) | 16 |
| Canton | (2) | 23 | Webster | (0) | 5 |
| Franklin | (0) | 11 | Worcester | (28) | 64 |
| Hull | (0) | 7 | Ludlow | (0) | 9 |
| Marlboro | (0) | 6 | Northampton | (0) | 13 |
| Milton | (2) | 16 | Prescott | (0) | 3 |
| Natick | (0) | 6 | Springfield | (16) | 93 |
| Needham | (0) | 19 | Greenfield | (8) | 66 |
| No. Attleboro | (0) | 7 | | | |

*The median endemic index is obtained by arranging in arithmetical sequence the monthly totals of reported cases for the past five years and selecting the middle figure. The numbers in parentheses after the name of each city and town indicate the median endemic index for that city or town; the numbers without parentheses indicate the cases reported during the current month.

### CONNECTICUT DEPARTMENT OF HEALTH

MORBIDITY REPORT FOR THE WEEK ENDING APRIL 17, 1926

| | | | |
|---|---|---|---|
| Diphtheria | 12 | Chickenpox | 44 |
| Last week | 17 | German measles | 17 |
| Diphtheria bacilli carriers | 5 | Influenza | 48 |
| Whooping cough | 91 | Mumps | 7 |
| Last week | 68 | Paratyphoid fever | 2 |
| Scarlet fever | 105 | Pneumonia, lobar | 114 |
| Last week | 91 | Tetanus | 1 |
| Measles | 460 | Tuberculosis, pulmonary | 28 |
| Last week | 595 | Tuberculosis, other forms | 2 |
| Bronchopneumonia | 84 | Gonorrhea | 8 |
| Cerebrospinal meningitis | 1 | Syphilis | 7 |

## NEWS ITEMS

ADDITION TO HARVARD FACULTY—Dr. Morton Prince, graduate of Harvard College in 1875, and, since 1912, professor of psychology, emeritus, of Tufts, will come to Harvard next September as associate professor of abnormal and dynamic psychology. Dr. Henry A. Murray, Jr., Harvard, '15, has been appointed research fellow in abnormal and dynamic psychology and will work with Dr. Prince. Dr. Murray was prominent as an undergraduate, rowing on three university crews and captaining that of 1915. After graduating from Harvard he studied at the College of Physicians and Surgeons in New York City, where he received the degree of doctor of medicine.

Dr. Prince received his doctor's degree at the Harvard Medical School in 1879, and was clinical instructor in diseases of the nervous system in the school from 1895 to 1898. In 1902 he became professor of nervous diseases in the Tufts Medical School, becoming emeritus in 1912. He was given an honorary LL.D. degree by Tufts in 1910. In that year also he was lecturer on abnormal psychology at the University of California.—*Boston Herald.*

DR. FREDERICK N. STEPHENS, of 390 Broadway, Winter Hill, Somerville, Mass., sailed Saturday, April 10, from New York, for several months' postgraduate study in Vienna, Austria, returning the first week of September.

DR. WILLIAM E. GYE HONORED—The London University grants a fellowship each year to one of its graduates who is believed to have done the greatest service toward the advancement of medical science.

Dr. William E. Gye is the winner of this fellowship for this year in recognition of his work on cancer.

The fellowship carries a grant of about $1500.

## REPORTS AND NOTICES OF MEETINGS

### ESSEX NORTH DISTRICT MEDICAL SOCIETY

BY invitation of the Trustees and Staff the Eighty-Fifth Annual Meeting will be held in Anna Jaques Hospital, Highland Ave., Newburyport, (Tel. 29) Wednesday, May 12, 1926.

Hospital open for inspection and clinical visits at 11 A. M. sharp and after meeting.

Attention Golfers: You are cordially invited to try your skill at Old Newbury Golf Club, on Newburyport Turnpike, near Dummer Academy (Tel. 8778-W). This course has a reputation of unusual interest. Come early and bring your sticks. Arrangements may be made with Roland L. Toppan, M.D., 148 High St., (Tel. 935,) or stay with us for a while after the meeting and make up a foursome.

Business meeting, 12 M. sharp.

Dinner, 1 P. M. sharp.

Papers, 2 P. M.

1. Heliotherapy in Surgical Tuberculosis, Zabdiel B. Adams, M.D., of Boston (20 minutes). Discussion by T. Raymond Healy, M.D., of Newburyport and Arthur B. Lyon, M.D., of Boston.

2. Symposium on Headache.

(*a*) W. Jason Mixter, M.D., of Boston (20 minutes).

(*b*) Channing Frothingham, M.D., of Boston (20 minutes).

Discussion by Leroy A. Schall, M.D., of Boston, John G. Jennings, M.D., of Boston, and Willis J. Bickford, Jr., D.M.D., of Newburyport. Discussion of all papers is open to those present.

Trustees, Staff and Superintendent are hoping for a large attendance. Hospital, City and Golf Club are yours for the day. Make it a real day and *come early.*

Note: Follow Haverhill-Newburyport trolley line, on High St. to Arlington St., and turn right. Hospital is on top of hill.

J. FORREST BURNHAM, M.D., *Secretary.*
567 Haverhill St., Lawrence, Mass.
RANDOLPH C. HURD, M.D., *President.*

### THE NORFOLK DISTRICT MEDICAL SOCIETY

THE Board of Censors of the Norfolk District Medical Society will hold a meeting for the examination of applicants for membership in the Massachusetts Medical Society at Roxbury Masonic Temple, 171 Warren St., on Thursday, May 6, at 4 P. M.

N.B.—Candidates must present their diplomas for examination by the censors.

FRANK S. CRUICKSHANK, M.D., *Sec.*,
23 Bay State Road.

## ESSEX NORTH DISTRICT MEDICAL SOCIETY

CENSORS' MEETING

THE censors of the Essex North District Medical Society will meet for the examination of candidates at the Hotel Bartlett, 53 Main St., Haverhill, Mass., (Telephone 8710) Thursday, May 6, 1926, at 2 o'clock sharp.

Candidates should make personal application to the secretary and present their medical diploma at least one week before the examination.

FORREST BURNHAM, *Secretary.*
567 Haverhill St., Lawrence, Mass.

## SUFFOLK DISTRICT MEDICAL SOCIETY

CENSORS' MEETING

THE censors of the Suffolk District Medical Society will meet for the examination of candidates at the Medical Library, No. 8 The Fenway, Thursday, May 6, 1926, at 4:00 o'clock.

Candidates should make personal application to the secretary and present their medical diploma at least one week before the examination.

ARTHUR H. CROSBIE, *Secretary.*
520 Commonwealth Avenue, Boston.

## ESSEX SOUTH DISTRICT MEDICAL SOCIETY

THURSDAY, May 6. Censors meet at Salem Hospital at 3:30 P. M. Candidates for examination should present their diplomas to the secretary at once.

Tuesday, May 11. Annual meeting at The Tavern, Gloucester. Dinner at 6:30 P. M.

Dr. Leo M. Davidoff will give an illustrated talk on "A Trip to the Far North with MacMillan."

Lady guests of members invited.

R. E. STONE, *Secretary.*
221 Cabot St., Beverly, Mass.

## TRUDEAU SOCIETY

THE next meeting of The Trudeau Society of Boston will be held on May 11, 1926, at 8:15 p. m., in Sprague Hall, The Medical Library, 8 The Fenway, Boston, Mass.

The paper will be by Dr. Edward O. Otis Subject—Symptomatic Treatment of Tuberculosis.

An active discussion is promised.

Physicians, Medical Students and Nurses are cordially invited.

GEORGE S. HILL, *Secretary.*

## BOOK REVIEWS

*Light and Health.* By M. LUCKIESH, Director, Lighting Research Laboratory, National Lamp Work of General Electric Co., and A. J. PACINI, Director, Department of Bio-physical Research, Victor X-ray Corporation of General Electric Co. Baltimore, The Williams and Wilkins Company, 1926.

In this comprehensive work the authors have delved considerably into the physics of light, touched a little on the bio-physics of the human race and the relation of light to life, and have attempted to evaluate so far as possible the effect of radiation on physiology and pathology. Although the raison d'etre of the volume is built up primarily about the ultra violet rays, the X-ray and diathermy are also given some attention.

As evidence that they are not filled with a boundless enthusiasm for the ultra-violet rays as a constant source of benefaction under all conditions, the dangers of over-irradiation from both natural and artificial sources are mentioned, and the failures of the rays in certain diseases are emphasized. Nevertheless the work as a whole is a panegyric of the rays. Important as they are and well recognized as their value is in definite conditions such as rickets and tuberculosis and perhaps in raising bodily resistance to other infections, it seems to me that their effect in other directions, such as on the glands of internal secretion, is not yet proven.

The mercury vapor quartz lamp, long in use, has only recently become popularized, and while all will recognize the disadvantages of lack of the ultra violet rays, the study of them has not yet reached a stage where their use can be recommended with unqualified enthusiasm in too great a range of conditions.

*The Pathology of Tumours.* By E. H. KETTLE, M.D., B.S. London. Second Edition. 285 pages, 159 illustrations. Published by Paul B. Hoeber, Inc., New York.

In this, the second edition of his Pathology of Tumours, Kettle has written a manual for students which aims to give the generally accepted views on this subject. The book is divided into three sections—The General Biology of Tumours, which includes their methods of dissemination and a resume of experimental work on cancer; The General Pathology of Tumours, and the Special Pathology of Tumours. The text, although concise, is sufficiently complete to convey a clear understanding of the subject. The illustrations are numerous and well selected. It is a book which is well suited for its purpose.

## SOCIETY MEETINGS

DISTRICT MEDICAL SOCIETIES

*Bristol South District Medical Society*

May 6, 1926—Annual meeting at New Bedford Public Library, 5 P. M.

*Essex South District Medical Society*

Thursday, May 6—Censors meet at Salem Hospital, 3:30 P. M.
Tuesday, May 11—The Tavern, Gloucester. Annual meeting. Speaker to be announced.

*Essex North District Medical Society*

May 12, 1926—The annual meeting at the Anna Jaques Hospital, Newburyport.

*Middlesex East District Society*

May—Annual meeting, Colonial Inn, North Reading. Speaker, Dr. E. H. Place. Subject to be announced.

*Notices of meetings* must reach the JOURNAL office on the Friday preceding the date of issue in which they are to appear.